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The Mediating Role of Depressive Symptoms in the Relationship Between Adverse Childhood Experiences and Cigarette Smoking

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The Mediating Role of Depressive Symptoms in the Relationship Between Adverse Childhood Experiences and Cigarette Smoking

by

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Thesis

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Master of Arts

The University of Texas at Austin

May, 2014

Abstract

The Mediating Role of Depressive Symptoms in the Relationship Between Adverse Childhood Experiences and Cigarette Smoking

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The University of Texas at Austin, 2014

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Adverse childhood experiences (ACEs), including various types of abuse and other forms of household dysfunction, have been consistently linked to increased rates of health risk behaviors and negative health outcomes in adulthood. Using data from the 2010 Centers for Disease Control and Prevention's (CDC) annual, nationwide Behavioral Risk Factor Surveillance System (BRFSS) telephone survey, this study tested whether self-reported symptoms of depression mediate the significant relationship between the number of ACEs an individual reports (expressed as an "ACE score") and whether they are a current or past smoker. A path model was produced using multiple regression, and indirect effects were tested using bootstrapping of 2000 samples. Results of analyses indicated that, among White, Asian, and Hispanic participants, self-reported depressive symptoms are indeed a significant, but only partial, mediator between participants' ACE score and their smoking status. These results suggest that for individuals of White, Hispanic, and Asian ethnicity, screening for a history of ACEs and treatment for depressive symptoms could increase the efficacy of smoking prevention efforts among youth and smoking cessation programs among adults. However, while depressive symptoms may explain some of the association between ACEs and smoking, these results suggest that other, unexamined factors also contribute to this pathway.

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Chapter 1: Introduction

The seminal Kaiser-CDC Adverse Childhood Experiences Study (ACE Study), conducted in the mid-1990s, established evidence of the widespread prevalence of stressful and traumatic events experienced during childhood among adults living in the United States. Adverse childhood experiences (ACEs) include physical, sexual, and emotional abuse, as well as exposure to other stressful family situations, including parental divorce, witnessing domestic violence, and having a household member who abuses drugs or alcohol, suffers from a mental illness, or is incarcerated. Data from a large sample of HMO members collected in the ACE study indicated that a history of these experiences is quite prevalent in the adult population, with more than 50% of adults reporting at least one ACE, and more than 13% reporting three or more of these experiences (Felitti et al., 1998).

Over the past twenty years, the critical contributing role of health risk behaviors such as smoking, alcohol use, and physical inactivity to mortality in the United States has been well documented (Mokdad, Marks, Stroup, & Gerberding, 2004). The ACE Study identified adverse childhood experiences as one important contributing factor to increased rates of health risk behaviors and negative physical health outcomes in adulthood (Felitti et al., 1998; Brown et al., 2009). Exposure to ACEs was found to dramatically increase rates of health risk behaviors, such as smoking and physical inactivity, and of diseases, including heart disease and cancer (Felitti et al., 1998). Moreover, ACEs appear to have a cumulative negative effect on health; the more ACEs an individual experiences, the higher his or her risk for a wide array of adverse health outcomes including substance abuse, depression, cardiovascular disease, cancer, diabetes, and early mortality (Felitti et al., 1998; Dong et al., 2004; Brown et al., 2009). This study introduced the use of the "ACE score", an integer count of the number of different

categories of adverse childhood experiences an individual reported, and established its strong and graded relationship to adverse behavioral and health outcomes.

Cigarette smoking is perhaps the most widespread health risk behavior in the United States, and is among those that has been most strongly linked to a history of ACEs. It is the leading cause of preventable death in the U.S., accounting for approximately 443,000, or one of every five, deaths in the country each year (Centers for Disease Control and Prevention, 2012). Smoking also results in an estimated $96 billion per year in direct medical costs and $97 billion per year in lost productivity (CDC, 2008). Smoking is estimated to increase the risk of developing coronary heart disease by 2 to 4 times, stroke by 2 to 4 times, lung cancer by 23 times for men and 13 times for women, and dying from chronic obstructive lung diseases (such as chronic bronchitis and emphysema) by 12 to 13 times (CDC, 2012). Despite these well-documented health outcomes, as of 2010 it was estimated that 19.3% of adults in the United States were cigarette smokers.

While the link between ACEs and adverse health outcomes in adulthood is well established, the pathway whereby ACEs lead to increased risk for health risk behaviors, diseases, and early mortality is not yet fully understood. The principal investigators of the ACE Study propose that ACEs lead to "social, emotional, and cognitive impairment," which in turn lead to the adoption of health risk behaviors, such as smoking, problem drinking, and overeating, all of which are known contributors to common diseases (Edwards, Anda, Gu, Dube, & Felitti, 2007, p. 6). Specifically regarding smoking, Anda and colleagues (2010) have proposed that the increased risk for smoking associated with a history of ACEs is mediated through depression; they describe smoking as a "logical, but probably unconscious, adaptation to depression" (Anda, Butchart, Felitti, & Brown, 2010, p. 94). Depression may indeed play a prominent role in this pathway, as it has been

shown to be both perhaps the most common long-term outcome of childhood trauma (Banyard, Williams, Saunders & Fitzgerald, 2008; Harper & Arias, 2004; Anda et al., 2007) and an important contributing factor to smoking initiation (Brook, Cohen, & Brook, 1998; Ferdinand, Blum, & Verhulst, 2001). However, the relationship between ACEs, depression, and smoking has not been directly tested (a) in a large, national sample of non-clinically referred adults or (b) using a continuous measure of depression symptomatology rather than a dichotomous measure of past or current depression.

This study will examine if and to what degree depressive symptoms mediate the relationship between adverse childhood experiences and smoking status. In 2009, the Centers for Disease Control and Prevention (CDC) included questions about ACEs in their annual, nationwide Behavioral Risk Factor Surveillance System (BRFSS) telephone survey, collecting data on demographic factors, health behaviors, and health status from a representative sample of the population in each state. Data from the four states that included both the optional ACEs module and an optional "Anxiety and Depression" module in their 2010 BRFSS surveys will allow for the examination of the relationship between ACEs, depressive symptoms, and smoking. Greater understanding of the role that depression and/or subclinical levels of depressive symptoms play in the relationship between ACEs and smoking can inform prevention and intervention efforts designed to eliminate the health disparities associated with adverse childhood experiences.

Chapter 2: Integrative Analysis

THE BIOPSYCHOSOCIAL MODEL

The biopsychosocial model of health posits that both psychological and physical illnesses result from a complex interplay of genetic, biological, psychological, behavioral, environmental, and social factors. This model rejects mind-body dualism, or the doctrine that mental processes and somatic processes are separate, instead acknowledging that mental and physiological processes affect one another and are both affected by an individual's environment (Engel, 1977). The biopsychosocial model is based on ecological systems theory (Bronfenbrenner, 1979), which locates the individual within hierarchically arranged social systems, beginning with the family system and extending outward through the overarching social and cultural environments in which an individual lives, all of which influence development and health through reciprocal and transactional processes (Engel, 1997).

The advent of the biopsychosocial model has led to a greater acknowledgement of the contributing role that psychosocial factors play in the etiology of disease (Suls & Rothman, 2004; Cohen, Janicki-Deverts, & Miller, 2007). For example, a large body of research has shown the detrimental effects of stress on the development of cardiovascular disease (Smith & Ruiz, 2002; Black & Garbutt, 2002), certain types of cancers (Antoni et al., 2006; Duijts, Zeegers, & Borne, 2003; Turner-Cobb, Sephton, & Speigel, 2001), immunity to infectious diseases (Segerstrom & Miller, 2004; Cohen & Williamson, 1991), autoimmune disease (Segerstrom & Miller, 2004; Ershler & Keller, 2000), and the progression of HIV to AIDS (Leserman et al., 2001; Cole, Kemeny, Fahey, Zack, & Naliboff, 2003). This research has shown that stress can lead to physical disease indirectly, via negative affective states and the adoption of unhealthy behaviors, and more directly, through alterations in physiological processes, such as the regulation of cortisol

secretion, which in turn affects immunological and inflammatory processes (Cohen, Janicki-Deverts, & Miller, 2007).

The leading causes of death in the U.S. throughout the past several decades—including heart disease, cancer, stroke, and diabetes—have all been shown to be critically influenced by human behaviors, such as smoking, alcohol use, physical inactivity, and obesity (Mokdad, 2002). In turn, individuals' likelihood of engaging in these behaviors has been linked to social and demographic factors, including poverty status, educational attainment, gender, and ethnicity (Huang, Hannon, Williams, & Harris, 2011; Garrett et al., 2011). Moreover, disparities in access to health care and the chronic stress associated with poverty and discrimination compound the adverse effects of health risk behaviors for ethnic minority and low socioeconomic status populations. For example, Latinos, African-Americans, and individuals living at or below the poverty line are significantly less likely to have health insurance than Whites or non-poor individuals, respectively (Moonesinghe, Zhu & Truman, 2011). Furthermore, research has shown that members of minority ethnic groups receive consistently lower medical care even when they are insured to the same degree and other determinants of health care, such as ability to pay for care, remain the same (Institute of Medicine, 2002). These factors help explain research findings that discrepancies in rates of health risk behaviors only explain a proportion of disparities in health outcomes between demographic groups (Huang et al., 2011).

Developmental Psychopathology and the Ecological-Transactional Model

Research conducted within the biopsychosocial model has demonstrated that chronic or extreme acute stress experienced at any point in the lifespan has the potential to detrimentally influence individuals' psychological, social, and physiological functioning (Cohen et al., 2007). Severe stress experienced during childhood, however, is

likely to have particularly damaging effects on individuals' functioning, since it has the potential to interfere with and disrupt normal developmental processes (Cicchetti & Lynch, 1993; Teicher, Andersen, Polcari, Andersen, & Navalta, 2002). Unlike theoretical perspectives that are characterized by efforts to isolate linear causal relationships between biological or environmental factors and specific manifestations of psychopathology, the developmental psychopathology perspective is characterized by multilevel analyses, which integrate evidence from various domains (genetics, clinical psychology, public health, etc.) and explore the interactions and transactions between biological, temperamental, and environmental factors in the development of both typical and atypical behavioral and emotional outcomes (Hinshaw, 2008). Drawing on recent research in epigenetics and neurobiology, developmental psychopathology acknowledges that throughout the course of an individual child's development, the child's environment continuously influences, and is in turn influenced by, genetic and biological factors (Caspi et al., 2002; Teicher et al., 2002). These external and internal processes interact in a reciprocal and transactional manner, driving both physiological and psychological development (Cicchetti, 2008).

Most research examining stress in childhood has focused on child maltreatment, which includes both abuse and neglect and represents perhaps the most conspicuous manifestation of an adverse developmental environment (Cicchetti, 2006). The ecological transactional model, which applies the developmental psychopathology perspective to childhood maltreatment, views traumatic stress experienced during childhood through a developmental lens, conceptualizing it as a potentiating factor for adverse developmental outcomes (Wilson, Hansen, & Li, 2011; Cicchetti & Lynch, 1993). It is the transactions among risk factors and compensatory (protective) factors in all levels of a child's environment (self, family, neighborhood, school) that determine both the risk for the

occurrence of maltreatment and the risk for adverse outcomes following maltreatment. In describing their model, Cicchetti and Lynch (1993) cite research that children who have experienced maltreatment display dysfunctional attachment relationships and representational models of the self and others, difficulties in emotional self-regulation, problematic peer relationships, and school difficulties. These "repeated developmental disruptions", they explain, interact with and build on one another to negatively alter maltreated children's developmental pathways (Cicchetti & Lynch, 1993, p. 109). This model calls for a consideration of the cumulative impact of various stressors in a child's life, since it is the balance between risk and compensatory factors that is considered predictive of adverse versus adaptive outcomes, rather than the mere presence or absence of a given risk factor (Cicchetti & Lynch, 1993; Sameroff, Seifer, Baldwin, & Baldwin, 1993). Because of this holistic conception of risk, the model also accounts for resilience, as it considers the compensatory effect that protective factors at all levels of a child's environment can have on developmental outcomes (Cicchetti & Lynch, 1993).

These related theoretical frameworks allow for a consideration of the immediate, intermediate, and long-term sequelae of adverse childhood experiences. Maltreatment and other ACEs and their consequent stress have been shown to result in disrupted attachments to caregivers, emotional dysregulation, disruptive behavior, and the development of negative core beliefs about the self, the world, and the future, all of which can negatively impact children's social adjustment and academic readiness (Wilson et al., 2011; Perry, 2008; Lumley & Harkness, 2007). In addition to these immediate behavioral and cognitive effects, traumatic stress may result in neurobiological changes, including diminished development of the left hemisphere (including the neocortex, hippocampus, and amygdala), reduced size of the corpus callosum, and attenuated activity in the cerebellar vermis (Teicher et al., 2002; Wilson et

al., 2011). These emotional, behavioral, social, and neurobiological effects may then alter subsequent development, contributing to or interacting with symptomatology that emerges during adolescence. Adolescents who have a history of maltreatment and/or other stressful childhood experiences are at increased risk for initiation of health risk behaviors, including substance use and risky sexual behavior, school problems, and internalizing and externalizing disorders (Lansford et al., 2002; Herrenkohl & Herrenkohl, 2007; Lester et al., 2009). In turn, these symptoms and behaviors predispose adolescents to maladaptive outcomes in adulthood, including substance abuse and depression (Chapman et al., 2004; Springer, Sheridan, Kuo, & Carnes, 2007). In this way, the effects of stress cascade through development, potentiating adverse outcomes in adulthood through a multitude of indirect pathways.

ADVERSE CHILDHOOD EXPERIENCES AND CIGARETTE SMOKING

As discussed above, despite its well-established and well-known relationship with diseases and death, cigarette smoking remains relatively common in the United States. Rates of smoking vary by gender, with 21.5% of men smoking compared to 17.3% of women. Smoking rates also vary significantly by ethnicity (with the highest rate among American Indians and lowest among Asians), level of education (with rates of smoking decreasing as highest level of education attained decreases), and socioeconomic status (with smoking more common among individuals living below the poverty line) (CDC, 2010; Garrett, Dube, Trosclair, Caraballo & Pechacek, 2011).

Given the persistence of smoking even in light of widespread information about its harmful long-term effects, it is important to understand why so many individuals continue to smoke. One factor that has been consistently associated with increased rates of smoking among adults is a history of adverse childhood experiences, including child maltreatment. Using data from the Kaiser-CDC ACE Study, Anda and colleagues (1999)

found that each category of ACE was significantly associated with ever smoking and with heavy smoking, and that there was a positive graded relationship between the number of ACEs an individual reported having experienced and their odds of being a smoker. Specifically, the authors found that adults who reported a history of more than five distinct categories of adverse childhood experiences had 5 times the odds of having started smoking at an early age, 3 times the odds of having ever smoked, and 2 times the odds of being a current smoker as those adults who reported no history of adverse childhood experiences (Anda et al., 1999). Similarly, studies using the same sample found that rates of hospitalization and use of medication for Chronic Obstructive Pulmonary Disease (COPD), and hospitalizations and death rates from lung cancer increased as the number of ACEs individuals reported increased (Anda et al., 1999; Brown et al., 2010). Ford and colleagues (2011) reached similar conclusions using more recent data from the 2009 Behavioral Risk Factor Surveillance System (BRFSS). They found that current smoking was significantly associated with each category of ACE, and that the prevalence of current smoking increased as the number of ACEs reported increased. Additionally, they found that participants reporting five or more ACEs were more than twice as likely to be current smokers as those reporting no ACEs (Ford et al., 2011).

These findings are supported by similar findings from both retrospective and longitudinal studies about the connection between maltreatment in childhood and smoking behavior in adulthood. Nichols and Harlow (2004) found that childhood sexual abuse, but not physical abuse, significantly increased the odds of smoking in adulthood. Similar results were obtained in a study of French adults, which found that 46% of adults who reported a history of sexual abuse were smokers, compared to 34% with no history of abuse. While this study included sexual abuse that occurred in adulthood as well as

during childhood, survivors of abuse that occurred in childhood were even more likely than survivors of abuse that occurred in adulthood to be smokers (57.1% compared to 40.4%), with survivors of childhood sexual abuse having 1.55 times the odds of being a smoker compared to their non-abused peers. Results from this study also showed that survivors of sexual abuse smoked more cigarettes per day and were less likely to quit smoking than smokers with no history of abuse (King, Guilbert, Ward, Arwidson, & Noubary, 2006). Data from the Chicago Longitudinal Study, comprised overwhelmingly of low-income, urban dwelling African-Americans, showed that childhood maltreatment (physical abuse, sexual abuse, and/or neglect) was associated with a 58.3% increase in the likelihood of daily smoking in young adulthood, resulting in a substantially higher rate of smoking among previously maltreated study participants than African-American adults in general (28.5% compared to 19.8%) (Topitzes, Mersky, & Reynolds, 2010).

The relationship between adverse childhood experiences and smoking has been suggested to result from symptoms of psychological distress, which lead to the adoption of smoking as a self-medication coping mechanism (Anda et al., 2010). A number of studies have supported the hypothesis that the relationship between traumatic childhood experiences and adult substance use, including cigarette smoking, may be partially mediated by psychological variables. In a study of more than 2,500 participants from various cities in the U.S., Douglas et al. (2010) examined the connection between childhood traumatic events, including violent crime, sexual and physical abuse, substance use in the household, and household instability (multiple caregivers and multiple relocations) and lifetime substance abuse disorders among adults (alcohol, cocaine, opioids, nicotine, cannabis, sedatives, and stimulants). They found that having a mood or anxiety disorder was a significant partial mediator between the number of these traumatic events experienced and risk of substance use disorders in adulthood. Specifically, the

odds ratio for having a substance abuse disorder declined from 1.76 to 1.42 with the inclusion of the dichotomous mood or anxiety disorder measure in the model (Douglas et al., 2010). Similarly, using data from a longitudinal study of Hispanic pre-adolescents, Fishbein and colleagues (2011) found that the effects of trauma on depression were established before adolescence and persisted into middle adolescence, and that depression mediated approximately 20% of the relationship between trauma and drug use initiation in middle adolescence (Fishbein, Novak, Krebs, Warner & Hammond, 2011).

These findings have been extended to smoking. Edwards and colleagues (2007) found that among participants in the Kaiser-CDC ACE Study, depression was a partial mediator of the relationship between the number of ACEs an individual reported and their odds of being a persistent smoker (continuing to smoke even after being diagnosed with a smoking related illness). Specifically, the inclusion of a dichotomous measure of past depression reduced the odds ratio of being a persistent smoker from 1.59 to 1.49 (Edwards et al., 2007). A recent prospective longitudinal study by Lewis et al. (2011) provided support for the causal link between childhood trauma, internalizing symptoms, and smoking. In this study of 522 primarily low-income youth, the researchers assessed history of maltreatment at age 12 based on both official Child Protective Services reports and youth self-report, internalizing symptoms (a composite of depression, anxiety, withdrawal, and somatic symptoms) at age 14, and smoking behavior at age 16. They found that after controlling for demographic factors, maltreated youth had significantly higher internalizing scores at age 14 and were nearly twice as likely to smoke at age 16 than non-maltreated youth. They found that internalizing symptoms at age 14 partially mediated the effect of maltreatment on smoking behavior, with the indirect effect of maltreatment on smoking via internalizing symptoms accounting for 8.9% of the relationship between maltreatment and smoking. Using data from the Chicago

Longitudinal Study, Topitzes et al. (2010) found that the significant link between child abuse and neglect and smoking was fully mediated by a comprehensive mediation model, which included social maladjustment, poor cognitive performance and/or school adjustment, and family instability. The authors found that depression and poor life satisfaction contributed mediating effects to the model, and that deficits in adolescent social-emotional skills were linked directly to daily smoking in adulthood. Based on these findings, they concluded that smoking may fulfill a compensatory or regulatory role in emotion management (Topitzes et al., 2010).

One notable limitation of both of the longitudinal studies referenced above is that they relied on a dichotomous classification of maltreated vs. non-maltreated youth and did not account for multiple trauma types or other forms of family dysfunction. Another limitation of many of these studies is their use of a dichotomous measure of past depression, rather than a measure of internalizing symptomatology that could consider the role of subclinical levels of symptoms in explaining the link between adverse childhood experiences and smoking.

In order for full or partial mediation to be plausible, the proposed mediator must be associated with both the presumed cause and the presumed effect (Baron & Kenny, 1986). Depressive symptoms have indeed been linked to both adverse childhood experiences on the one hand and smoking on the other hand. They are thus plausible potential mediators of the well-documented relationship between childhood adversity and trauma and tobacco and alcohol use in adulthood.

Adverse Childhood Experiences

With the exception of the studies utilizing the data collected in the Kaiser-CDC ACE Study, most research examining the impact of adverse childhood experiences either examines one of these experiences in isolation or uses a dichotomous classification of

maltreatment or abuse. Large bodies of research have explored the immediate and long-term effects of maltreatment during childhood, including physical, sexual and emotional abuse and physical and emotional neglect. Other research has investigated the impact of the other common adverse childhood experiences, including divorce and parental substance abuse, on children's emotional, social, and behavioral functioning, once again typically examining these experiences in isolation from one another. A review of this literature illustrates the wide array of behavioral and emotional problems that all of these experiences have been linked to during childhood, adolescence, and adulthood, including virtually every DSM-IV disorder, from ADHD to depressive disorders and post-traumatic stress disorder (PTSD) (Perry, 2008). Consistent with the biopsychosocial, developmental psychopathology, and ecological-transactional models, recent research, including that associated with the ACE Study, has begun to consider the interrelations between these categories of adverse experiences and to conceptualize them as contributing in a cumulative fashion to long-term outcomes.

Child Maltreatment: Immediate and Long-term Effects

A large body of research has documented the long-lasting adverse effects of individual forms of child maltreatment on children's emotional, behavioral, and social functioning. Maltreatment includes both acts of commission (i.e. abuse) and acts of omission (i.e. neglect) by parents or caregivers that result in harm or threat of harm (Gilbert et al., 2009). Maltreatment includes: physical abuse, typically defined as the use of physical force or implements against a child that causes or has the potential to cause physical injury; sexual abuse, which includes any completed or attempted sexual act, sexual contact, or non-contact sexual interaction with a child by an adult; psychological or emotional abuse, defined as intentional behavior that conveys to a child that he/she is worthless, flawed, unloved, unwanted, endangered, or valued only in meeting another's

needs; and physical and/or emotional neglect, which is the failure to meet a child's basic physical, emotional, medical, or educational needs, to provide adequate nutrition, hygiene, or shelter, or to ensure a child's safety (Gilbert et al., 2009). Witnessing interparental violence is also considered a form of child maltreatment, given its association with other forms of abuse and neglect and its well-documented ability to provoke traumatic stress in children (Moylan et al., 2010). The research detailing the effects of each individual type of maltreatment will be briefly summarized below.

Physical abuse during childhood has been associated with increased rates of depression and aggression in children (Johnson et al., 2002; Herrenkohl & Herrenkohl, 2007) and decreased rates of prosocial behavior (Prino & Peyrot, 1994). Adolescents who were physically abused as children display higher levels of depression, anxiety, social problems, and aggression than their non-abused peers (Lansford et al., 2002; Springer et al., 2007) and are at higher risk for suicidal thoughts and behaviors (Evans, Hawton & Rodham, 2005). These adverse effects persist into adulthood; adults who were physically abused display higher levels of depression and suicidal behavior than adults who were not maltreated as children (Brown, Cohen, Johnson & Smailes, 1999; Chapman et al., 2004).

Psychological or emotional abuse has also been associated with adverse psychological outcomes in adolescence and adulthood. Adolescents who report having experienced psychological or emotional abuse as children report higher levels of non-suicidal self-injury than their non-abused peers (Buser & Hackney, 2012). Childhood psychological abuse has also been associated with depression, anger, and low self-esteem in adulthood (Harper & Arias, 2004; Briere & Runtz, 1990). Results from the ACE Study showed that women who reported psychological abuse as children were 2.7 times as

likely to report lifetime depression and 3.1 times as likely to report recent depression as women who did not report emotional abuse (Chapman et al., 2004).

Childhood sexual abuse has been linked with internalizing and externalizing symptoms in adolescence (Herrenkohl & Herrenkohl, 2007) and increased lifetime risk for PTSD, anxiety disorders, depression, suicidal behavior, eating disorders, sexual risk behavior, and academic difficulties (Paolucci, Genuis & Violato, 2001; Chen et al., 2004; Evans et al., 2005). Research has found that these associations remain statistically significant even after accounting for contextual factors such as socioeconomic status and family functioning (Fergusson, Boden & Horwood, 2008; Brown et al., 1999). Adult victims of childhood sexual abuse also report higher levels of substance abuse disorders and of certain physical health problems than non-victims, including sleep disorders, poor general health, gastrointestinal disorders, gynecological disorders, chronic pain, cardiopulmonary problems and obesity (Chen et al., 2004; Leserman, 2005; Irish, Kobayashi, & Delahanty, 2010; Simpson & Miller, 2002). Survivors of childhood sexual abuse are also more likely to be sexually re-victimized in adulthood (Arriola, Louden, Doldren, & Fortenberry, 2005).

Although neglect is the category of maltreatment most frequently recorded by child protective agencies, it has been the subject of considerably less research than other forms of child maltreatment, likely because, as an act of omission, it is more difficult to specifically define and document than abuse (Gilbert et al., 2009). Studies of children removed from neglectful situations have demonstrated that severe neglect has profoundly negative impacts on young children, causing serious developmental, intellectual and social delays (Read, Perry, Moskowitz, & Connolly, 2001; Perry, 2008). Neglected children show elevated levels of depression and social withdrawal (Prino & Peyrot, 1994), deficits in emotion knowledge (Sullivan, Carmody & Lewis, 2010), and

internalizing problems in adolescence (Herrenkohl & Herrenkohl, 2007). Unfortunately, questions about neglect were not included in the 2010 BRFSS Adverse Childhood Experiences module, and thus neglect is not included as one of the ACEs in the present study.

Witnessing interparental or domestic violence in the home has been documented to be traumatic for children and frequently co-occurs with other forms of maltreatment (Moylan et al., 2010). Children who report having witnessed interparental violence report more symptoms of depression and anxiety than children never exposed to violence at home and are more likely to be rated by parents and teachers as being physically aggressive and having more behavior problems in general than their non-exposed peers (Moylan et al., 2010; McFarlane, Groff, O'Brien, & Watson, 2003; Evans, Davies, & DiLillo, 2008; Sternberg et al., 1993). Additionally, children exposed to domestic violence frequently have elevated scores on scales of post-traumatic stress disorder and often meet criteria for the disorder (Lehmann, 1997; Kilpatrick & Williams, 1998).

Other Forms of Family Dysfunction: Immediate and Long-term Effects

Child maltreatment often occurs in the context of other forms of family dysfunction, including divorce, household substance abuse, household mental illness, and parental incarceration (Ammerman, Kolko, Kirisci, Blackson, & Dawes, 1999; Freisthler, 2011; Phillips, Erkanli, Keeler, Costello, & Angold, 2006). Each of these factors has been independently associated with negative impacts on children's emotional and behavioral functioning. Similar to the research on child maltreatment, most of the research considering the incidence and impact of these experiences has examined them in isolation. This research will be briefly summarized here.

There is a significant body of research documenting the effects of divorce on children (see Amato, 2001; Amato, 2010). Research indicates that children of divorced

parents score significantly lower than children of continuously married parents on a number of measures of academic achievement, behavior, psychological adjustment, self-concept, and social relations (Amato, 2001). However, these associations may be largely explained by inter-parental hostility, rather than the divorce itself (Baxter, Weston, & Qu, 2011). Nonetheless, the negative impacts of divorce seem to persist into adult for some individuals. Longitudinal research has shown that, for women, parental divorce is negatively associated with the quality of parent-child relationships, self-esteem, and satisfaction with social support (Mustonen, Huurre, Haukkala, Kiviruusu, & Aro, 2011). Adults whose parents divorced have lower levels of psychological well being, report more problems in their own marriages, and are at greater risk of getting divorced themselves (Amato, 2010).

Parental substance abuse has been associated with increased emotional and behavior problems in children and greater risk-taking behaviors in adolescents, which may be partially mediated by problematic parenting practices resulting from substance abuse (Lester et al., 2009). Notably, research has demonstrated that parents with past or current substance abuse problems may be more likely to physically abuse their children than parents with no such disorder (Ammerman et al., 1999; Freisthler, 2011). Children of parents with substance abuse disorders, including alcoholism, have been found to have higher rates of internalizing symptoms, depression, conduct disorder, and overall behavior problems, as well as lower global functioning scores, than children whose parents do not abuse substances (Merikangas, Dierker, & Szatmari, 1998; Chatterji & Markowitz, 2001; Christensen & Bilenberg, 2000). Adolescents whose parents abuse substances are significantly more likely to smoke, use alcohol, and use illicit drugs than children of control families (Merikangas et al., 1998; Lester et al., 2009), though it is not clear how much of this association is due to heritable factors (Brown, 2008). Parents'

history of substance abuse has also been found to be positively associated with the severity of their adult offspring's own substance abuse (Boyd, Plemons, Schwartz, Johnson, & Pickens, 1999).

Parental mental illness is linked to increased risk for psychological problems in children, adolescents, and adults. Parental depression has been consistently associated with internalizing and externalizing symptoms in children and with parent-child conflict (Beck, 1999; Kane & Garber, 2004). Adults reporting that they grew up with a mentally ill household member are more than twice as likely to report having ever had a depressive disorder and nearly 3 times as likely to report a current depressive disorder (Chapman et al., 2004). Possible causal explanations for this link include genetic transmission, observational learning from exposure to depressive symptomatology, and impaired parenting behavior (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Furthermore, research suggests that parents with mental illness may be more likely to maltreat their children, as the rates of psychiatric disorders, including mood disorders, substance use disorders, and personality disorders are elevated among parents of maltreated children (DeBellis et al., 2001; Famularo, Kinscherff, & Fenton, 1992; Ammerman et al., 1999).

The vast majority of research on familial incarceration focuses on the impacts of parental incarceration. Research has suggested that children's home lives are often severely disrupted by their parent's incarceration, as it commonly results in little or no contact with the incarcerated parent, confusion and uncertainty about the future, feelings of grief and ambiguous loss, moves to other households and/or schools, and increased stress and financial difficulties incurred by remaining caregivers (Aaron & Dallaire, 2010; Bocknek, Sanderson, & Britner, 2009; Dallaire & Aaron, 2010; Hairston, 2010). Adolescents with incarcerated parents are significantly more likely to be suspended or expelled or to fail or drop out of school than their peers who have not experienced

parental incarceration (Hanlon et al., 2005; Trice & Brewster, 2004). Much of the increased risk for delinquency and school problems is likely due to parental incarceration typically occurring within the context of other sociodemographic risk factors; children with histories of parental incarceration experience elevated rates of poverty, parental substance abuse, parental mental illness, harsh and punitive parenting, and exposure to domestic violence (Phillips, Burns, Wagner, Kramer, & Robbins, 2004; Phillips et al., 2006).

The ACE Score: Interrelatedness and Cumulative Impact

Given research findings that have documented that individuals' risk for reporting depression and for smoking increase as the number of categories of adverse childhood experiences reported increases, this study will utilize an ACE score for each participant. The "ACE score", which is an integer count of the number of categories of ACEs to which an individual was exposed during childhood, was first used in the Kaiser-CDC ACE Study. The use of this score in the present study is based on two related factors. First, research has shown that different categories of maltreatment and family dysfunction are highly interrelated and tend to co-occur. Second, in line with the developmental psychopathology and ecological-transactional approaches, the cumulative impact of stressful experiences is likely to be more predictive of outcomes than the individual experiences themselves. Findings from research using the ACE Study sample and other diverse samples support the use of a cumulative experience score based on both of these considerations.

Interrelatedness of different categories of adverse childhood experiences

Results from the ACE study demonstrated that the different categories of adverse childhood experiences are highly interrelated (Dong et al., 2004; Felitti et al., 1998).

Using a subsample of 8,629 participants from the ACE study, Dong et al. (2004) found that each of the 10 categories of experiences they examined (physical abuse, emotional abuse, sexual abuse, exposure to domestic violence, parental divorce or separation, household mental illness, household substance abuse, physical neglect, emotional neglect, and incarceration of a household member) was statistically significantly associated with each other category. If a person reported that they had experienced any one category of ACE, they were 2 to 18 times more likely to report another category than persons reporting no ACEs. Furthermore, among persons reporting any single ACE category, 86.5% reported at least one other, while 52% reporting at least three more (Dong et al., 2004).

Other research supports the finding that different types of traumatic childhood experiences are highly interrelated. High rates of co-occurrence have been found among all types of child maltreatment, with most studies examining co-occurrence finding that most children exposed to one type are exposed to at least one other (Saunders, 2003), and that each type of maltreatment is significantly correlated with each other type (Herrenkohl & Herrenkohl, 2007; Ney, Fung & Wickett, 1994). The co-occurrence of domestic violence and child physical abuse is well documented (Appel & Holden, 1998; Sternberg et al., 1993; Moylan et al., 2010; Sternberg, Lamb, Guterman, & Abbot, 2006), and husband to wife aggression has been associated with child abuse potential for both fathers and mothers (Margolin, Gordis, Medina, & Oliver, 2003). Co-occurrence rates for child physical and/or sexual abuse and domestic violence of between 32% and 80% have been found in meta-analyses (Appel & Holden, 1998) and longitudinal studies (Saunders, 2003; Springer et al., 2007). High rates of co-occurrence between physical abuse and psychological abuse have also been found, with studies reporting that more than 90% of

cases of physical maltreatment also include psychological maltreatment (Clausen & Crittenden, 1991; Ney et al., 1994; Downs, Capshew, & Brindels, 2004).

Research has also demonstrated that various types of maltreatment are associated with other forms of household dysfunction. Data from the Wisconsin Longitudinal Study showed that childhood physical abuse was significantly associated with elevated rates of parental drinking problems and serious marital problems between parents (Springer et al., 2007). Similarly, results from the National Comorbidity Survey showed that sexual abuse was associated with all other childhood adversities examined, including verbal abuse, physical abuse, witnessing domestic violence, parental mental illness, and parental substance abuse. Furthermore, victims of childhood rape or molestation were likely to report a number of other adversities; among these victims, the most commonly reported number of adversities was five or more (Molnar, Buka, & Kessler, 2001).

These diverse research findings demonstrate that most types of adverse childhood experiences do not occur in isolation, but rather as constellations of risk in children's lives. Thus, when considering the impact of childhood trauma or risk factors such as divorce or parental incarceration, it is important to assess for and consider related adverse childhood experiences.

The cumulative impact of multiple ACEs

The ecological transactional framework considers the sequelae of childhood trauma as dependent upon the transactions among vulnerability and protective factors at all levels of a child's environment. Cicchetti and Lynch (1993) argue that "an increased presence of enduring vulnerability factors and transient challengers" in a child's ecology results in higher risk for problematic developmental processes and outcomes (p. 99). This framework thus argues for the consideration of the full array of adverse conditions and

experiences present in childhood, rather than a dichotomous or categorical classification of maltreatment or trauma.

The ACE Study found a strong graded relationship between adverse childhood experiences and negative health outcomes in adulthood. Specifically, as the number of different categories of adverse childhood experiences an individual reported increased, his or her odds of engaging in a wide array of health risk behaviors, of having depression, of having ever attempted suicide, and of having a number of diseases, including cancer, stroke, and diabetes, also increased. This relationship was particularly notable for depression; individuals reporting two ACEs were 2.4 times as likely to report current depression than those reporting no ACEs and those reporting four or more ACEs were 4.6 times as likely to report current depression. Notably, the most dramatic relationship observed was between ACEs and having ever attempted suicide. Participants with two ACEs were 3 times as likely to have ever attempted suicide as those reporting no ACEs, while those reporting four or more ACEs were 12.2 times as likely (Felitti et al., 1998).

Other research has found similar graded relationships between traumatic experiences or childhood adversities and negative psychological and behavioral outcomes in adulthood. In a study based on a large national telephone survey of women, victims of both childhood sexual and physical abuse were found to be more than twice as likely to have a chronic disabling mental health condition than victims of just one of these types of maltreatment. Dual victims were also more likely to report having used drugs in the previous month than victims of one type of maltreatment (Thompson, Arias, Basile & Desai, 2002). These results were similar to those found in a large survey of randomly selected female HMO members in Seattle, which found that the number of childhood maltreatment categories individuals endorsed was significantly associated with higher odds of mental disability status, as well as with a greater number of reported physical

symptoms and physician-recorded diagnoses. Reporting multiple types of childhood maltreatment was also associated with engaging in a greater number of health risk behaviors, including smoking, alcoholism, and having sex with a partner before knowing their sexual history (Walker et al., 1999). Likewise, Nichols and Harlow (2004) found that women reporting both childhood sexual abuse and childhood physical abuse were 3.5 times more likely to be smokers than women reporting no history of abuse, compared to those reporting just one type of abuse, who were only 1.4 times as likely to be smokers.

Similarly, longitudinal research examining the impacts of physical abuse and exposure to domestic violence during childhood on psychological functioning in adolescence has found that dual exposure to these forms of violence is predictive of a wider array of both internalizing and externalizing problems than exposure to either alone, and that adolescents exposed to both score higher on measures of delinquency and depression than those exposed to either alone (Moylan et al., 2010). However, other studies have found only limited or mixed support for a greater negative effect on children's functioning of combined domestic violence and physical abuse than either alone, finding that children exposed to either or to both are not significantly different from one another on measures of psychological functioning (Sternberg et al., 2006; Kitzmann, Gaylord, Holt, & Kenny, 2003).

Studies including a wider array of types of trauma and adversity have consistently found cumulative negative impacts of multiple kinds of exposures. In a large community survey, Turner and Lloyd (1995) found that the number of lifetime traumas adults had experienced (including eight childhood experiences) was associated with significant graded increases in rates of major depression, rates of substance abuse disorders, and depression symptomatology scores. Focusing specifically on child maltreatment, Ney et al. (1994) found that a combination of physical abuse, physical neglect and verbal abuse

was most strongly associated with a reported lack of enjoyment in living, belief that one's own future will be poor, and belief that one has a poor chance of having a happy marriage (Ney et al., 1994).

More recent studies have examined the cumulative impact on children's functioning of victimization in home, school, and community contexts. Using a nationally representative sample of youth, ages 2-17, Finkelhor and colleagues (2007) found that "poly-victimization", referring to the experience of multiple kinds of victimization in home, school, and community contexts, was a powerful predictor of trauma symptoms. Specifically, they found that poly-victims made up 80% of the 10-17 year olds with clinically significant levels of anxiety symptoms and 86% of those with clinically significant levels of depression symptoms in their sample (Finkelhor, Ormrod, & Turner, 2007). Similarly, Holt and colleagues (2007) found that fifth grade students classified as "multiple victims" based on their having been exposed to victimization in peer, family, and community contexts reported significantly higher psychological distress, greater social difficulties, lower grades, and higher rates of sexual victimization than their peers who were either minimally victimized or victimized only in the peer context (Holt, Finkelhor, & Kantor, 2007).

Furthermore, Finkelhor et al. (2007) found that the inclusion of a poly-victimization measure in their analyses either eliminated or greatly reduced the predictive power of individual types of victimization, suggesting that the experience of multiple kinds of victimizations was a stronger determinant of negative functioning than the individual experiences themselves. Likewise, they found that poly-victims had significantly higher symptom scores than children who reported chronic victimization of one type, further suggesting that multiple *different* victimization experiences are more deleterious to psychological functioning than even chronic victimization. The authors did

note one exception to this pattern; victims of chronic maltreatment at home had particularly high depression scores, even compared to poly-victims, a finding that indicates that maltreatment in the home context may have a uniquely detrimental impact on youth's psychological functioning (Finkelhor et al., 2007). Similarly, Herrenkohl and Herrenkohl (2007) found that a general construct of child maltreatment is a stronger predictor of both internalizing and externalizing symptoms among children than any individual kind of maltreatment or related social and family stressors. However, they noted that physical and sexual abuse did appear to have unique predictive value over and above the general construct of maltreatment, again suggesting that while a general measure of adversity or trauma may be the most powerful predictor of negative psychological functioning, these types of abuse may be particularly damaging (Herrenkohl & Herrenkohl, 2007).

The use of a score that captures the cumulative impact of multiple forms of adverse experiences is supported by the work of Sameroff and colleagues (1993) who demonstrated that a multiple risk score was more important in predicting children's IQ than any single environmental risk factor, and that the multiple risk score remained a significant predictor of IQ even after the strongest individual predictors were controlled for. Moreover, the researchers found that the multiple risk score was important in longitudinal prediction of children's IQ, even when their previously measured IQ was taken into account. They also found that for families with more than three risk factors, the pattern of risks was less important than the total number of risks (Sameroff et al., 1993). These research findings strongly suggest that the use of a cumulative risk score, such as the ACE score used in the present research, is likely to be a better predictor of adverse outcomes than the individual categories of adverse childhood experiences themselves. Furthermore, when individual types of trauma, violence, or family dysfunction are

examined in isolation without considering other co-occurring types, the impact of whichever individual factor is being examined is likely to be exaggerated (Finkelhor et al., 2007).

Path A: ACEs to Depressive Symptoms

Across types of adverse childhood experiences, one of the most consistently noted long-term psychological impacts is depression. Research shows that the diverse short and mid-term impacts of traumatic stress experienced during childhood, including behavior problems, substance use, and internalizing problems, are all associated with increased risk for depression in adulthood (Lahey, 2008; Klein, Torpey, Bufferd & Dyson, 2008). Using data from the ACE study, Chapman et al. (2004) found that the ACE score had a significant graded relationship to both lifetime prevalence of depressive disorders and recent depression. Women reporting three or more ACEs were 3 times more likely than those reporting no ACEs to report ever having had a depressive disorder, while women reporting five or more ACEs were 5 times more likely than those with no ACEs to report lifetime prevalence of depression and more than 6 times more likely to report a recent depressive disorder. The relationship was weaker but still significant for men; men reporting four or more ACEs were more than twice as likely as men reporting none to have a lifetime history of depression (Chapman et al., 2004).

These findings have been supported by other studies documenting increased risk for depression among adults who were maltreated as children. In a longitudinal study that followed children with documented cases of maltreatment into adulthood, Widom and colleagues (2007) found that adults who experienced physical abuse or multiple types of abuse were at an increased risk for lifetime major depressive disorder (MDD) compared to matched controls. Consistent with research showing a graded relationship between trauma exposure and negative outcomes, participants who had experienced more than one

type of abuse were 1.75 times more likely than controls to experience MDD, compared to participants reporting a history of physical abuse only, who were 1.59 times as likely as controls to have MDD (Widom, DuMont, & Czaja, 2007). In a review of studies examining the impact of early maltreatment on later functioning, Harkness and Lumley (2008) concluded that past maltreatment led to a two- to five-fold increase in the risk for later depressive disorders.

The mechanisms whereby adverse childhood experiences lead to negative psychological outcomes are varied. Children's normal neural and emotional development depends on consistent nutrition, appropriate sensory input, and warm and dependable attention from caregivers. The impact of being deprived of any of these essential environmental inputs, as happens in cases of maltreatment and chaotic home environments, can be wide-ranging and pervasive. Recent research in neuroscience has demonstrated that early stress is capable of actually altering children's developing neurobiological systems, resulting in lasting biological changes (Perry, 2008). Research in genetics and epigenetics has shown that gene expression is not as predetermined as once thought, and that the way genes are expressed can actually be altered by a child's environment (Anda et al., 2010). For example, Caspi and colleagues (2003) found that polymorphisms in the promoter region of the serotonin transporter gene (5-HTTLPR) moderate the effect of stressful life events, including childhood maltreatment, on adult depression. Childhood maltreatment increased risk for depression in early adulthood among individuals with the common short allele but not with the common long allele (Caspi et al., 2003).

As described above, maltreatment, traumatic stress, and other forms of adversity may impact development through neurobiological, socialization, cognitive, or emotional processes. One well-established pathway between childhood adversity and depressive

symptomatology is through the formation of cognitive schemas. The cognitive theory of depression states that negative core beliefs or schemas about the self, the world, or the future lead to depression by influencing how one perceives and interprets their day-to-day experiences (Beck, 1967, 1976). Research suggests that individuals who experience depression have more negative content in their beliefs about the self, world, and future (i.e. "I am worthless", "I am helpless") and a cognitive organization that engenders easier activation of negative beliefs than positive beliefs, referred to as depressotypic cognitive organization (Lumley & Harkness, 2007; Dozois & Beck, 2008; Dozois 2007; Dozois, 2002; Segal, Hood, Shaw, & Higgins, 1988). Schema theory, developed by Young and colleagues, proposes that early maltreatment can lead to the development of early maladaptive schemas, such as vulnerability to harm, unlovability, or worthlessness, that when consolidated and strengthened over childhood, eventually lead to negative core beliefs and depressotypic cognitive organization (Young & Brown, 1994; Young, Klosko, & Weishaar, 2003).

Recent research supports these hypotheses. Specifically, in a study of depressed adolescents, Lumley and Harkness (2007) found that physical abuse and emotional maltreatment were significantly associated with the presence of early maladaptive schemas, including failure and vulnerability, which were in turn associated with symptoms of anxiety and anhedonia. Furthermore, they found that beliefs of emotional deprivation, which are related to worthlessness, mediated the relationship between physical abuse and depressive symptoms, while beliefs of social isolation and self-sacrifice mediated the relationship between emotional maltreatment and symptoms. In another study, Lumley and Harkness (2009) found that higher levels of self-reported childhood parental emotional maltreatment and physical abuse (but not sexual abuse) were associated with depressotypic cognitive organization in a sample of college

students, and that the relationship between childhood maltreatment and current or past depression was mediated by depressotypic cognitive organization. Similarly, Browne and Winkelman (2007) found that the relationship between childhood maltreatment and trauma symptoms was mediated by cognitive distortions in individuals' beliefs about safety, controllability, and internal attribution for events. Results from a study of college students suggested that childhood emotional maltreatment, but not physical or sexual abuse, were significantly related to cognitive vulnerability to depression, levels of hopelessness, and depression, and that depressotypic cognitive style fully mediated the relation between levels of reported childhood emotional maltreatment and hopelessness depression, a type of depression characterized by high levels of cognitions about hopelessness (Gibb et al., 2001). Consistent with the biopsychosocial model, these diverse bodies of research have demonstrated that stressful experiences during childhood can affect individuals both psychologically and physiologically, through complex and interdependent pathways.

Path B: Depressive Symptoms to Smoking

A large body of literature has shown that negative affective states, particularly depressive symptoms, are associated with initiation of smoking (Brook et al., 1998; Brown, Lewinsohn, Seeley, & Wagner, 1996; Escobedo, Reddy, & Giovino, 1998; Ferdinand et al., 2001), increase in smoking behavior and transition from recreational to addictive smoking (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; McKenzie, Olsson, Jorm, Romaniuk, & Patton, 2010; Schleicher, Harris, Catley, & Nazir, 2009), and degree of nicotine dependence among smokers (Lerman et al., 1996). While some studies have found a negative association between depressive symptoms and smoking cessation (Anda et al., 1990; Salive and Blazer, 1993; Glassman et al., 1988; Covey, Glassman,

Stetner and Becker, 1993), other studies have found no association between depression and rates of cessation (Breslau et al., 1998).

The mechanisms whereby depressive symptoms influence smoking behavior are undoubtedly multiple; however, one pathway that has been supported by extensive empirical research is that of self-medication, or negative affect reduction. The self-medication hypothesis proposes that smoking is used to regulate and alleviate depressive symptoms (Repetto, Caldwell, & Zimmerman, 2005). Chaiton and colleagues (2010) directly tested the self-medication hypothesis in a longitudinal study of adolescents. They found that the perceived self-medication value of smoking was indeed significantly positively associated with depressive symptoms, and that participants who smoked more than 25 cigarettes per week had consistently higher self-medication scores than those who smoked less (Chaiton, Cohen, O'Loughlin, & Rehm, 2010).

Related to the self-medication hypothesis is the proposition that individuals are more likely to smoke if they hold positive outcome expectancies about smoking (Friedman-Wheeler, Ahrens, Haaga, McIntosh, & Thorndike, 2007). Outcome expectancies are estimates that a certain behavior will precipitate a particular outcome (Bandura, 1977). In the case of smoking, positive outcome expectancies include the belief that smoking will reduce negative affect, provide sensory stimulation, or facilitate social interaction. Schleicher and colleagues (2009) found support for this hypothesis in a study of undergraduate students. The authors found that higher depressive symptoms significantly predicted a higher number of cigarettes smoked in the previous month, and that this relationship was fully mediated by participants' self-reported negative affect regulation expectancies about smoking (Schleicher et al., 2009). Similarly, McChargue and colleagues (2004) found in a sample of undergraduate students that those with a history of depression were more than 5 times as likely to be current smokers as those with

no history of depression, and that the relationship between past depression and smoking status was fully mediated by participants' positive reinforcement expectancies about smoking (McChargue, Spring, Cook, & Neumann, 2004). Among current smokers, Lerman and colleagues (1996) found that smokers with higher scores on a measure of depressive symptoms scored significantly higher than non-depressed smokers on a measure of negative affect reduction expectancies about smoking (Lerman et al., 1996). In two samples of adults who smoked cigarettes daily, one seeking treatment and one not, Friedman-Wheeler and colleagues (2007) found that individuals with more self-reported depressive symptoms and proneness to depression had stronger positive outcome expectancies about smoking. The authors postulated that such positive outcome expectancies about smoking may lead depression-prone smokers to engage in fewer alternative non-smoking coping behaviors and thus be more dependent on smoking as a coping mechanism (Friedman-Wheeler et al., 2007).

Audrain and colleagues (2010) found support for this hypothesis in a longitudinal study of young adult smokers, the results of which demonstrated that the effect of depressive symptoms on smoking was fully mediated by participants' use of substitute reinforcers, such as pleasant events and enjoyable activities. Specifically, the authors found that higher symptoms of depression in emerging adulthood predicted declines in non-smoking alternative reinforcers, which, in turn, predicted increases in smoking uptake or rate in young adulthood. Importantly, they found that this relationship held even for individuals with subclinical levels of depression (Audrain-McGovern, Rodriguez, Rodgers, & Cuevas, 2010).

STATEMENT OF PURPOSE

Adverse childhood experiences, including various forms of abuse and other common types of household dysfunction (i.e. divorce, parental mental illness, parental

substance abuse), have been identified as critical predisposing factors for the development of health risk behaviors in adulthood and, in turn, heightened risk for a wide array of diseases and consequent early mortality. Cigarette smoking is one of the most hazardous health risk behaviors common in the United States, leading to more than 440,000 deaths in this country each year. Like other health risk behaviors, smoking has been linked to ACEs. However, while the relationship between ACEs and health risk behaviors has been well documented, the pathway linking them is not fully understood. Investigators have proposed that ACEs lead to impairments in emotional and social functioning, which then lead to the adoption of health risk behaviors. Depression has been identified as common outcome of ACEs and as predisposing factor for a number of health risk behaviors, including smoking. Using a large sample of adults from four geographically and demographically diverse U.S. states, this study will test whether self-reported symptoms of depression account for a significant proportion of the relationship between the number of ACEs individuals report and their smoking status in adulthood.

Chapter 3: Methods

Research Questions and Hypotheses

Research Question 1: In a large community sample of randomly selected adults, is there a significant relationship between the number of adverse childhood experiences (ACEs) an individual reports (expressed as an integer ACE score) and his or her self-reported smoking status (current heavy, current occasional, former, or never smoker)?

Hypothesis 1: It is hypothesized that there will be a statistically significant relationship between the ACE score and self-reported smoking status.

Rationale: Previous research using a large sample of HMO members has established that the ACE score is a significant predictor of whether an individual is a current smoker (Felitti et al., 1998), a current heavy smoker (Anda et al., 1999), a former smoker, or a persistent smoker (Edwards et al., 2007). These results were replicated using data collected by the CDC in 2009 from five U.S. states (Ford et al., 2011). These findings are supported by other studies demonstrating significant links between childhood sexual abuse (Nichols & Harlow, 2004; King et al., 2006) or a general measure of maltreatment (Topitzes et al., 2010) and smoking.

Research Question 2: Does the relationship between the ACE score, depressive symptoms, and smoking status vary based on ethnicity, gender, or socioeconomic status, each of which are independently associated with each variable?

Hypothesis 2: It is hypothesized that the relationship between the ACE score, depressive symptoms, and smoking status does not vary based on the measured demographic variables.

Rationale*:* Rates of reported ACEs, depression, and smoking have been shown to vary by ethnicity, gender, and socioeconomic status. However, no research has

demonstrated that the relationship between these variables is *moderated* by any of these demographic factors.

Research Question 3: Are self-reported symptoms of depression a significant mediator of the relationship between the ACE score and smoking status?

Hypothesis 3: It is hypothesized that depressive symptoms significantly, but only partially, mediate the relationship between the ACE score and smoking status.

Rationale: Previous research has established that a dichotomous measure of past or current depression significantly but only partially mediates the relationship between the ACE score and smoking status (Edwards et al., 2007). The continuous measure of depressive symptoms used in this study may be a more sensitive measure of the emotional distress that is hypothesized as the pathway between ACEs and smoking, and thus, it is expected that it will account for a significant proportion of the total effect of the ACE score on smoking status.

METHODS

Participants

This study utilized data from the Center for Disease Control and Prevention's 2010 annual Behavioral Risk Factor Surveillance System (BRFSS) telephone survey, which is made available to the public on the CDC's website (Centers for Disease Control and Prevention, 2010a). The BRFSS is a state-based system of health surveys that collects information on health risk behaviors, preventive health practices, and health care access primarily related to chronic disease and injury. It is conducted annually in all 50 states, the District of Columbia, Puerto Rico, the U.S. Virgin Islands, and Guam. Data are collected on a monthly basis by each state or region's Department of Health. More than 350,000 adults are surveyed each year for the BRFSS (CDC, 2006). Information about

the BRFSS and downloadable data files are available at the CDC's website at http://www.cdc.gov/brfss/.

Data were included from only the four states that administered both the "Adverse Childhood Experiences" and "Anxiety and Depression" optional modules of the BRFSS. These states were Hawaii, Nevada, Vermont, and Wisconsin (CDC, 2010b). The total sample size from these four states was 20,711. The Council of American Survey Research Organizations (CASRO) response rates for these four states were 49.14%, 50.71%, 60.48%, and 57.82%, respectively. The CASRO rate is defined as the proportion of all eligible respondents in the sample for whom an interview has been completed. CASRO rates of at least 40% are considered acceptable (CDC, 2004, 2011a). The percentage of females and of non-Hispanic whites, and the mean age of respondents in the sample, organized by state, is included in Table 1 (CDC, 2011a).

Instruments

Behavioral Risk Factor Surveillance System Questionnaire

The BRFSS survey is administered over the phone by trained interviewers. In each state, an independent probability sample from non-institutionalized adults aged 18 years and older with telephones is selected using disproportionate stratified sampling (CDC, 2006). Data collection is the responsibility of each state health department. State health departments are trained by the CDC, and report all data to the CDC once it is collected. The BRFSS survey includes core sections, which all states must administer, asking about demographics, general health status, health care access, common chronic diseases, and health-related behaviors (including health risk behaviors such as smoking, and preventative health behaviors such as cancer screenings). Each year the BRFSS also includes a number of optional modules, which individual states decide whether or not to

administer (CDC, 2006). The core portion of the survey lasts an average of 18 minutes (CDC, 2011b).

Many questions included in the BRFSS are taken from established national surveys, such as the National Health Interview Survey or the National Health and Nutrition Examination Survey. Prior to their inclusion in the survey, any proposed new questions go through testing with focus groups in order to ensure that potential respondents understand them correctly and can provide accurate answers (CDC, 2010c). Results from the BRFSS have been shown to be substantially similar to results from the National Health Interview Survey, which is conducted via within-home, in-person interviews (Nelson, Powell-Griner, Town, & Kovar, 2003).

Validity and reliability of the BRFSS

Most questions on the BRFSS have been determined to be of at least moderate reliability and validity and many have been determined to be highly reliable and valid (Nelson, Holtzman, Bolen, Stanwyck, & Mack, 2001). Research examining the reliability of demographic variables on the BRFSS has uniformly found them to be highly reliable, with reliability coefficients of 0.92 - 1.00 for age, 0.96 - 1.00 for sex, and 0.87 - 0.97 for race/ethnicity. Studies examining the reliability of measures of socioeconomic status have found test-retest correlations of 0.70 - 0.92 for educational attainment and 0.86 - 0.97 for income. There are no studies examining the validity of demographic measures on the BRFSS, but studies examining the validity of demographic variables in similar samples have concluded that they typically have high validity (Nelson et al., 2001).

Smoking module

A number of studies have examined the reliability and validity of smoking measures on the BRFSS and have found them to be high and consistent. The reliability

has been found to be high for ever smoking and current smoking (κ = 0.79 – 0.94 and κ = 0.83 – 1.00, respectively), and slightly lower for former smoking (κ = .58 – 0.86). Studies examining the validity of smoking questions have found that compared to biochemical measures, they have a sensitivity of 78% for men and 86% for women, and a specificity of 97% for men and 96% for women. Prevalence estimates for current, ever, and past smoking measures on the BRFSS have been found to consistently be within 1 to 3 percentage points of prevalence estimates produced from biochemical measures and in-person interviews (Nelson et al., 2001).

Adverse Childhood Experiences module

The BRFSS "Adverse Childhood Experiences" module included a total of 11 questions that were grouped into 8 categories of abuse or household dysfunction (CDC, 2010b). All questions in the module were based on questions used in the large-scale Kaiser-CDC ACE Study (Anda et al., 2010). Before being included for the first time in the BRFSS survey in 2009, the questions were piloted with focus groups, a procedure to which all new BRFSS questions are subjected. As a result, the wording of questions differs slightly from the questions in the Kaiser-CDC Study (CDC, 2010b). No studies have yet examined the validity or reliability of the BRFSS ACE module.

Questions in the Kaiser-CDC Study about psychological and physical abuse and violence against the respondent's mother were taken from the Conflicts Tactics Scale. Four questions from Wyatt (1985) were adapted to assess sexual abuse. Questions about exposure to drug or alcohol abuse were adapted from the 1988 National Health Interview Survey (Felitti et al., 1998). Additional questions were developed to ask about five other categories of household dysfunction, including witnessing domestic violence (Straus & Gelles, 1990); exposure to substance abusing (Schoenborn, 1995); mentally ill household

members; parental separation or divorce; or having an incarcerated household member (Anda et al., 1999).

All of the categories of abuse and household dysfunction used in the Kaiser-CDC ACE Study and the overall ACE score have been established to have good test-retest reliability as defined by Fleiss (1981) and moderate to substantial test-retest reliability as defined by Landis and Koch (1977) (Dube, Williamson, Thompson, Felitti, & Anda, 2004). The kappa coefficients for each category are: .66, 95% CI [.55 – .76] for emotional abuse; .55 [.47 – .63] for physical abuse; .69 [.61 – .77] for sexual abuse; .75 [.68 – .81] for household substance abuse; .77 [.68 – .85] for witnessing domestic violence; .51 [.42 – .61] for household mental illness; .46 [.27 – .65] for an incarcerated family member; and .86 [.81 – .91] for parental separation or divorce. The adjusted kappa for the overall ACE score was .64, 95% CI [.36 – .60] (Dube et al., 2004).

Anxiety and Depression module

The "Anxiety and Depression" module included a total of 10 questions asking about the frequency of symptoms of depression and/or anxiety and whether or not respondents had ever been diagnosed with an anxiety disorder or a depressive disorder (CDC, 2010b). Since the module did not include questions about all of the symptoms of anxiety, only the questions related to depression were considered in the present study. This module is based on the eight-item Patient Health Questionnaire (PHQ-8) depression scale, which consists of eight of the nine criteria on which the DSM-IV diagnosis of depressive disorders is based (Kroenke et al., 2009; American Psychiatric Association, 1994). The ninth item of the PHQ-9, which assesses the final DSM-IV criteria, suicidal or self-injurious thoughts, was not included in the BRFSS because telephone interviewers would not be able to provide adequate intervention if this symptom were to be reported by respondents. Previous research shows that this item is the least likely to be endorsed of

all nine items on the PHQ-9, and that its deletion has only a minor effect on scoring of the measure (Kroenke et al., 2009; Huang, Chung, Kroenke, Delucchi, & Spitzer, 2006; Kroenke & Spitzer, 2002; Lee, Schulberg, Raue, & Kroenke, 2007; Rief, Nanke, Klaiberg, & Braehler, 2004). The question response set was changed to standardize it with other BRFSS questions by asking the number of days in the past 2 weeks the respondent had experienced each symptom (Kroenke et al., 2009).

Validity and reliability of PHQ-8

No studies have yet examined the Anxiety and Depression module of the BRFSS, but a number of studies have investigated the reliability and validity of the measure on which it was based. The PHQ-9 has been shown to be valid and reliable as both a diagnostic and severity measure useful in both clinical and population settings, including when administered by telephone (Kroenke et al., 2009; Pinto-Meza, Serrano-Blanco, Penarrubia, Blanco, & Haro, 2005). It has been found effective for detecting depression in diverse racial and ethnic groups and among individuals with a wide array of physical health conditions (Kroenke et al., 2009; Huang et al., 2006). In comparisons of the PHQ-8 and PHQ-9, the two versions of the measure have been shown to have almost identical sensitivity, specificity, and positive predictive value for depression (Kroenke & Spitzer, 2002).

The diagnostic validity of the PHQ-9 was established by comparing it to results from a structured mental health professional (MHP) interview. Using the MHP re-interview as the criterion standard, a PHQ-9 score of 10 or greater (on a scale of 0 – 27) had a sensitivity of 88% and a specificity of 88% for major depression (Kroenke, Spitzer, & Williams, 2001). The internal consistency of the PHQ-9 as established in the initial validation studies was high, with a Cronbach's α of .89 when administered in a primary care setting and .86 in an OB-GYN clinic. Test-retest reliability of the PHQ-9 was also

high. The correlation between the PHQ-9 measure completed by patients in the clinic and the measure administered over the phone by a mental health professional within 48 hours was .84, with nearly identical mean scores for the two conditions (5.08 vs. 5.03) (Kroenke et al., 2001).

Variables

ACE score

Following the methodology established in the Kaiser-CDC ACE Study, an "ACE score" was summed for each participant representing the number of categories of adverse childhood experiences they reported, ranging from 0 to 8 (Felitti et. al, 1998). The questions, response options, and criterion for each category of ACE are included in Appendix 3. Because the BRFSS did not include questions about physical or emotional neglect, the maximum ACE score for participants was 8, rather than 9, as in the ACE Study.

Depressive symptoms

Depressive symptoms were analyzed in a continuous fashion using the scoring criteria established for the PHQ-8. Based on the methodology used by Kroenke et al. (2009), the modified response set for the eight depressive symptoms questions was converted back into the response set of the PHQ-8, with 0 to 1 day = "not at all," 2 to 6 days = "several days," 7 to 11 days = "more than half the days," and 12 to 14 days = "nearly every day," and points (0 to 3) assigned to each category, respectively. The points for each item were then summed to produce a total score for each participant between 0 and 24 points. On the PHQ-8, a total score of 5 to 9 represents mild depressive symptoms; 10 to 14, moderate symptoms; 15 to 19, moderately severe symptoms; and 20

to 24, severe symptoms (Kroenke et al., 2001). This total score was used as the continuous measure of depressive symptoms.

Smoking status

Participants' smoking status was analyzed using a four-level calculated variable included in the BRFSS dataset, computed from participants' responses to four individual questions about tobacco use (CDC, 2010d). The original calculated variable (1 = current smoker, now smokes everyday, 2 = current smoker, now smokes some days, 3 = former smoker, 4 = never smoked) was transformed in order to have a positive, rather than negative, correlation with the ACE score and depression symptoms variables (0 = never smoked, 1 = former smoker, 2 = current smoker, now smokes some days, 3 = current smoker, now smokes everyday).

Demographics

Given their established association with the variables of interest, race/ethnicity, age, gender, and socioeconomic status were used as control variables in analyses. Socioeconomic status was analyzed using a composite variable of self-reported income level (the BRFSS uses an eight level annual income level variable ranging from "less than $10,000" to "more than $75,0000") and self-reported educational attainment (the BRFSS uses a six level educational attainment measure ranging from "never attended school or only attended kindergarten" to "college, 4 years or more") (CDC, 2010d). Using the entire four state sample, the Pearson correlation between these two variables was computed to be statistically significant ($r = .399$, $p < .001$). Both variables were converted to *z*-scores and then averaged to produce the composite socioeconomic status variable. Ethnicity was analyzed using a computed eight level variable having the following categories: White only, Black only, Asian only, Native Hawaiian or other

Pacific Islander only, American Indian or Alaskan Native only, Other race only, Multiracial, and Hispanic (CDC, 2010d).

Chapter 4: Results

Analytic Plan

First, zero order correlations were computed among the variables of interest. In order to test hypothesis 1, a multiple regression was conducted to evaluate the association between the ACE score and smoking status, with demographic variables entered as control variables, including seven dummy variables for each ethnicity category. Next, hypothesis 2 was tested in order to determine whether the sample could be analyzed as a whole for the test of hypothesis 3 or should be divided into subsamples based on demographic characteristics. In order to test for interaction effects by demographic variables, each demographic variable was centered and then interaction terms were created by multiplying each centered demographic variable by the centered ACE score variable. In order to test for interactions by ethnicity, interaction terms were created for each dummy variable. These interaction terms were separately entered as the final step in sequential regressions with the ACE score and all demographic variables as predictors, and first smoking status, then depressive symptoms (the proposed mediator variable), as the outcome variable. Change in the adjusted r-squared statistic was examined in order to determine whether an interaction was present (Keith, 2006). Finally, in order to test hypothesis 3, path models were conducted using sequential and a series of simultaneous multiple regressions testing the direct and indirect effects of the ACE score, control demographic variables, and depressive symptoms on smoking status. All analyses were computed using the statistical software SPSS version 16. Bootstrapping with 2000 samples was used for testing the significance of the mediated effects and to produce bias-corrected percentile confidence intervals (Preacher & Hayes, 2004, 2008). Although the CDC typically applies weighting to BRFSS data, data in the present study were not weighted since the research questions do not involve prevalence estimates.

PRELIMINARY ANALYSES

Pearson correlations among all of the variables used in the models are included in Table 2, excepting ethnicity, which was not included because it is a non-dichotomous categorical variable. Consistent with previous research and with the hypotheses of this study, among the entire four state sample, all of the variables of interest were significantly related to each other and to the demographic variables. Both the ACE score and depressive symptoms were significantly negatively correlated with age, significantly positively correlated with female sex, and significantly negatively correlated with socioeconomic status. Smoking status (being a current or former smoker) was negatively correlated with age, negatively correlated with female sex, and negatively correlated with socioeconomic status. The ACE score, depressive symptoms, and smoking status were all positively correlated with each other.

Table 3 shows the prevalence of each ACE score and the mean ACE score by demographic categories. Consistent with previous research, the mean number of ACEs reported was greater for women (1.42) than for men (1.28) and greater for respondents age 34 and younger than for those 35 and older. Among ethnic categories, participants of Asian ethnicity reported the lowest mean ACE score (.63) and American Indian/Alaskan Native participants reported the highest (2.28).

Table 4 includes the mean PHQ-8 score, representing depressive symptoms, for participants by demographic categories. Consistent with research on depression, the mean score was higher for women (3.26) than for men (2.66), and higher among participants younger than 55. Among ethnic groups, participants of Asian ethnicity had the lowest mean score (2.11), while American Indian/Alaskan Natives and Blacks had the highest and second highest mean scores, respectively (5.52 and 4.61).

Table 5 presents the percentage of individuals in demographic categories who reported each smoking status. Consistent with national statistics, more males than females reported being current or past smokers (54.2% vs. 44.5%), and the highest rates of current smoking and ever smoking were reported by participants of American Indian or Alaskan Native ethnicity (35.3% and 65.9%), while the lowest were reported by participants of Asian ethnicity (8.3% and 35.1%).

Among age groups, the highest rate of current smokers (everyday or some days) was among participants ages 25-34 (24.8%), while the lowest rate was among participants age 65 and older (8.5%). The highest rates of former smokers, however, and of ever smokers (former or current), were among participants age 65 and older (45.5% and 54.1%, respectively), while the lowest rate of ever smokers was among participants age 18 to 24 (24.2%).

PRIMARY RESULTS

Hypothesis 1: Relationship between ACEs and Smoking Status

The overall multiple regression for smoking status, using the entire four state sample, was statistically significant (R^2 = .086, F[11, 17160] = 147.614, p < .001). All of the predictor variables together accounted for 8.6% of the variance in participants' self-reported smoking status. Socioeconomic status, gender, age, and the dummy variables associated with Asian, American Indian, and Hispanic ethnicities each had a statistically significant effect on smoking status (see Table 6 for full regression results). Participants' ACE scores, the variable of interest, also had a statistically significant effect on smoking status (b = .088, β = .158, t [17160] = 20.756, p < .001). These results indicate that even after accounting for demographic characteristics associated with smoking behavior, the ACE score does have a significant effect on smoking status in adulthood.

Hypothesis 2: Interaction Effects

No significant interaction effects were found for gender, age, or socioeconomic status with either smoking status or depressive symptoms as the outcome variable. A significant interaction effect was found for race/ethnicity with depressive symptoms as the outcome variable ($\Delta R^2 = .002$, $F[7, 16488] = 5.623$, $p < .001$). Given the presumed heterogeneity of the "Other" and "Multiracial" categories, participants in these categories were not included in subsequent analyses. Examination of the coefficients associated with the interaction terms revealed that the significant interaction was caused by the dummy variables associated with the Black, Native Hawaiian, and American Indian groups. Because the sample sizes of the Native Hawaiian and American Indian groups were relatively much smaller than those of the other ethnic groups ($N = 151$ and $N = 145$, respectively), these ethnic groups were not analyzed in the present study. Because the sample size for the Black group was large ($N = 443$), a separate analysis was performed for this group. The remaining groups were combined (White, Asian, and Hispanic) and the dummy variables associated with Asian and Hispanic ethnicity were used to control for ethnicity in subsequent analyses. Because of the large size of these samples, coefficients were considered statistically significant only if they were significant at the $p < .01$ level.

Hypothesis 3: The Mediating Role of Depressive Symptoms in the Relationship Between ACEs and Smoking Status

White, Asian, and Hispanic participants

Among White, Asian, and Hispanic participants, the overall multiple regression for smoking status including all predictor variables was statistically significant ($R^2 = .088$, $F[7, 14326] = 197.808$, $p < .001$). All of the predictor variables together accounted for 8.8% of the variance in participants' self-reported smoking status. Of the control

variables, socioeconomic status, gender, and the dummy variables associated with Asian and Hispanic ethnicity were all significant predictors of participants' smoking status; age was not. Both of the variables of interest, the ACE score and depressive symptoms, also had significant effects on smoking status. Of the demographic variables, sex, age, and socioeconomic status all had significant direct effects on participants' ACE scores and on depressive symptoms. Asian ethnicity was a significant predictor of the ACE score, but not of depressive symptoms, while Hispanic ethnicity was not a significant predictor of either the ACE score or depressive symptoms. Results of the simultaneous and sequential regressions used in these analyses are included in tables 7 – 10.

After accounting for sociodemographic characteristics, depressive symptoms mediated a significant proportion of the variance in smoking status accounted for by the ACE score. Specifically, the standardized indirect effect of the ACE score on smoking status through depressive symptoms was computed to be $\beta = .025$. This indirect effect represents 16.1% of the total effect of the ACE score on smoking status. Using the normal theory based Sobel test, this indirect effect was found to be statistically significant ($z = 10.892$, $p < .001$). Furthermore, the bias-corrected 95% confidence interval for the unstandardized indirect effect obtained by bootstrapping did not include 0 ($b = .0145$, 95% CI [.0113 - .0179]). Table 11 shows the total, indirect, and direct standardized effects of each of the variables of interest and each of the control variables on participants' smoking status.

Black participants

Among Black participants, the overall multiple regression for smoking status including all predictor variables was statistically significant ($R^2 = .086$, $F[5, 438] = 8.213$, $p < .001$). All of the predictor variables together accounted for 8.6% of the variance in participants' self-reported smoking status. All of the demographic variables

(age, sex, and socioeconomic status) were significant predictors of smoking status (see Table 12). However, for this subsample, after controlling for demographic factors, neither the ACE score nor depressive symptoms had significant direct or total effects on smoking status, and thus, no mediation analysis was performed.

Although neither the ACE score nor depressive symptoms were significant predictors of Black participants' smoking status, the ACE score was nonetheless significantly associated with depressive symptoms among Black participants. The overall multiple regression for depressive symptoms was statistically significant ($R^2 = .193$, $F[4, 439] = 26.276$, $p < .001$), and all of the predictor variables together accounted for 19.3% of the variance in participants' depressive symptoms. Age was not a significant predictor of depressive symptoms, but sex, socioeconomic status, and ACE score were (see Table 14 for full regression results). Of these variables, the ACE score had the strongest effect on depressive symptoms ($b = .936$, $\beta = .325$, $p < .001$). Interestingly, in contrast to the results for the White, Asian, and Hispanic subsample and previous research, sex did not have a significant effect on the ACE score among Black participants. Consistent with results from the other subsample and existing research, both socioeconomic status and age did have significant effects on the ACE score (see Table 15 for full regression results).

Chapter 4: Discussion

This study explored the associations between adverse childhood experiences, depressive symptoms, and smoking status in adulthood. As predicted, among all participants, the number of adverse childhood experiences reported (expressed as an integer "ACE score") was significantly and positively associated with both smoking behavior and depressive symptoms in adulthood. Preliminary analyses indicated a significant interaction between ethnicity (specifically Black vs. non-Black) and the ACE score in predicting depressive symptoms, and thus, Black participants were analyzed separately from White, Asian, and Hispanic participants.

Among the White, Asian, and Hispanic subgroup, the ACE score had a significant effect on smoking status, and depressive symptoms were found to mediate a significant proportion of this total effect. These results suggest that for White, Asian, and Hispanic adults, symptoms of depression do indeed help explain some of the pathway between exposure to ACEs in childhood and smoking in adulthood. However, the indirect effect through depression accounted for just 16.1% of the total effect of ACEs on smoking among White, Asian, and Hispanic participants.

These findings suggest that other unaccounted for factors explain a majority of the relationship between ACEs and smoking among these ethnic groups. Other research suggests that these may include lack of parental support or monitoring, peer influence, and the neurobiological effects of trauma, which may predispose individuals to be particularly susceptible to nicotine's addictive properties (Topitzes et al., 2010; Wilson et al., 2011; Pomerleau & Pomerleau, 1987).

Interestingly, these results were not replicated among the subsample of Black participants. Among Black participants, after accounting for sociodemographic characteristics, neither the ACE score nor depressive symptoms had a significant effect

on smoking status. These results suggest that previous research linking childhood exposure to ACEs to smoking behavior in adulthood may not generalize to Black Americans. Among this subsample, ACEs were indeed significantly associated with depressive symptoms in adulthood. However, this association did not appear to lead to smoking behavior.

These results also suggest that different factors may be associated with exposure to ACEs among Black Americans. While female sex has been consistently associated with a higher ACE score in previous research—a finding that was replicated among the White, Asian, and Hispanic participants in this study—among Black participants, sex did not have a significant effect on the number of ACEs reported. This result suggests that the gender disparity in reported ACEs may not generalize to Black Americans. This finding suggests that either exposure to ACEs is more evenly distributed among males and females of Black ethnicity, and/or that Black males are more willing to report ACEs than non-Black males.

Both SES and age were associated with the ACE score for both subgroups; for all participants, higher SES and older age were associated with a lower ACE score. As other research has indicated, the association between age and the ACE score suggests that ACEs have been on the rise throughout the previous generation, which is certainly the case for certain ACE categories such as divorce (Amato, 2010) and parental incarceration (Glaze & Maruschak, 2008; Hairston, 2010). Additionally, these results suggest that younger participants may be more likely to report ACEs than older participants.

LIMITATIONS

The most significant limitation of this study was the use of cross-sectional, retrospective data. Because the measure of depressive symptoms was collected at the same time as the measure of smoking status, it is not possible to conclude that the

symptoms of depression preceded participants' smoking behavior. However, past research has shown that depression symptomatology is relatively stable over the life course (Lovibond, 1998; Foley, Neale & Kendler, 2001). A large NIMH study found that individuals who at one time meet criteria for Major Depressive Disorder were symptomatic approximately 60% of the time with at least subthreshold levels of depressive symptoms (Judd, Schettler, & Akiskal, 2002). Moreover, there is strong evidence suggesting that exposure to childhood trauma is one of the most significant risk factors for the development of chronic depression in adulthood (Riso, Miyatake, & Thase, 2002). Furthermore, prior research indicates that there is a causal relationship between depression and smoking. A number of longitudinal studies have established that symptoms of depression precede the initiation of smoking and the transition to heavy smoking (McKenzie et al., 2010; Audrain-McGovern et al., 2010; Repetto et al., 2005). The few studies that have found smoking behavior to precede the onset of depression have considered only the diagnostic category of Major Depressive Disorder (Breslau et al., 1998; Brown et al., 1996), or the dichotomous classification of "depressed" (Wu & Anthony, 1999), not taking into account that subclinical levels of depressive symptoms may have been present before smoking initiation.

A related limitation is the retrospective nature of the report of adverse childhood experiences. Because of the lapse in time between the experiences and the time of the survey, some adults may have difficulty recalling the experiences (Della Famina, Yeager, & Lewis, 1990). Moreover, the stressful nature of these experiences may cause memory impairments (Wilson et al., 2011), or because of the potential stigma and psychological discomfort associated with recalling and reporting these experiences, individuals may choose to deny events that they did experience. However, all of these factors would be expected to result in *underestimates* of the actual occurrence of ACEs, a phenomenon

that has been observed in studies comparing retrospective reports of abuse during adulthood to documentation of abuse made in childhood (Della Famina et al., 1990; Williams, 1995; Hardt & Rutter, 2004). Furthermore, other methods of assessing exposure to ACEs such as abuse, including parent report, contemporaneous child report, and official records (i.e. police reports, child protective services documentation) present both methodological and ethical challenges, and have all been found to result in even greater underestimates of actual prevalence than retrospective self-report (Gilbert et al., 2008).

Another limitation of this study is the use of a measure of depressive symptoms only, rather than a broader measure of internalizing symptoms assessing both depression and anxiety. On the one hand, symptoms of depression and anxiety are highly correlated and often co-occur (Löwe et al., 2008; Kessler et al., 2003). On the other hand, each disorder is associated with unique impacts on psychosocial functioning (Löwe et al., 2008), while comorbid depression and anxiety are associated with greater symptom severity and impairment than either disorder alone (Cyranowski et al., 2012). Like depression, anxiety has been shown to be both a common outcome of ACEs (Afifi, Enns, Cox, Asmundson, Stein, & Sareen, 2008) and a predisposing factor for smoking (Kassel, Stroud, & Paronis, 2003). Thus, a broader construct of internalizing symptoms may account for more of the relationship between ACEs and smoking status.

An additional limitation of this study is the use of a measure of participants' socioeconomic status based on their own level of educational attainment and reported income, rather than of the socioeconomic status of their families when they were children, which would be expected to be a more robust predictor of exposure to ACEs. However, socioeconomic status during childhood is correlated with socioeconomic status

during adulthood, since individuals from families of higher income are more likely to have access to educational opportunities and, consequently, higher paying jobs.

A final limitation of this study is the skew of the sample towards older, White Americans. The large size, geographic diversity, and non-clinically referred nature of the sample is undoubtedly one of its primary strengths, and allowed for the testing of interactions by demographic variables. However, the relatively smaller size of the subsamples of ethnic minority groups limit the generalizability of the findings related to these groups.

IMPLICATIONS AND FUTURE DIRECTIONS

These results suggest that among Americans of White, Asian, and Hispanic ethnicity symptoms of depression do indeed help account for some of the association between adverse childhood experiences and smoking status, providing some support for the proposed pathway through emotional distress between ACEs and health risk behaviors in adulthood. These results have several implications for the prevention and treatment of smoking. They suggest that screening and treating adolescents and young adults for a history of adverse childhood experiences and even subclinical levels of depression may help prevent the adoption of smoking or the transition to heavy smoking. They also suggest that screening and treatment for depressive symptomatology may help adult smokers with a history of adverse childhood experiences successfully quit smoking.

However, the finding that depressive symptoms mediate only a proportion of the association between ACEs and smoking suggests that for many individuals, the adoption of health risk behaviors such as smoking may not result from directly perceived symptoms of distress, even at subclinical levels. Instead, for some individuals, ACEs may co-occur with or contribute to the presence of other predisposing factors for health risk behaviors, such as parental use of these behaviors, associations with peers who practice

these behaviors, or neurobiological alterations that increase susceptibility to the addictive properties of substances. Alternatively, the use of cross-sectional data in this study may have resulted in an underestimate of the effect of depressive symptomatology on the adoption of smoking as a coping mechanism. Since depressive symptoms were measured at the same time as smoking status, it is possible that some individuals who may have had depressive symptomatology at the time of their smoking initiation no longer had these symptoms when surveyed. If this possibility were supported by longitudinal evidence, it would suggest that individuals exposed to ACEs may find improvement in their emotional functioning over time, but may persist in habitual behaviors adopted earlier that continue to compromise their health and well-being. In order to explore both of these possibilities, longitudinal studies should be conducted that measure adolescents' history of ACEs, emotional functioning, peer associations, and substance use over time.

Although the 2010 BRFSS did not include questions assessing for all the symptoms of anxiety, it did include two questions asking whether participants had ever been diagnosed with or told by a doctor that they had either a) a depressive disorder, or b) an anxiety disorder. Participants' responses to these two questions could be used to construct more comprehensive mediation models using logistic regression, including both depression and anxiety and accounting for remitted cases of these disorders. However, the use of these dichotomous measures would be less powerful statistically than the use of the continuous measure of depressive symptoms used in the present study, and would not account for subclinical levels of depressive or anxious symptomatology.

As discussed above, although previous longitudinal research supports the causal pathway from depressive symptoms to cigarette smoking, some research does support a bidirectional relationship between depressive symptoms and smoking behavior. The use of structural equation modeling with the data used in the present study would permit

testing if a model allowing for a bidirectional relationship between depressive symptoms and smoking status provides a better fit for the data than the model used in the present study.

Finally, perhaps the most significant finding of this study was the finding that ethnicity moderated the relationship between ACEs, depressive symptoms, and health risk behaviors. Previous studies examining the relationship between ACEs and health risk behaviors have not considered ethnic differences (Felitti et al., 1998, Anda et al., 1999, Anda et al., 2007; Brown et al., 2009). The finding that after controlling for demographic factors, ACEs were not associated with smoking behavior among Black participants indicates that more research needs to be conducted using larger and more geographically diverse samples of Black Americans in order to determine whether ACEs are associated with this particular health risk behavior among this demographic group. Moreover, it suggests that the findings regarding the relationships between ACEs and health behaviors and outcomes concluded from predominantly White samples may not be generalizable to Black Americans. More generally, it suggests that research investigating the relationship between adverse childhood experiences, emotional distress, and health risk behaviors should consider the potential differential effects ACEs may have on the emotional and behavioral functioning of different ethnic groups. Given that interaction effects were also found for the American Indian/Alaskan Native and Native Hawaiian groups, it would be interesting to analyze the associations between the variables of interest among these groups as well.

Appendix A: Figures

Figure 1: Proposed Path Model

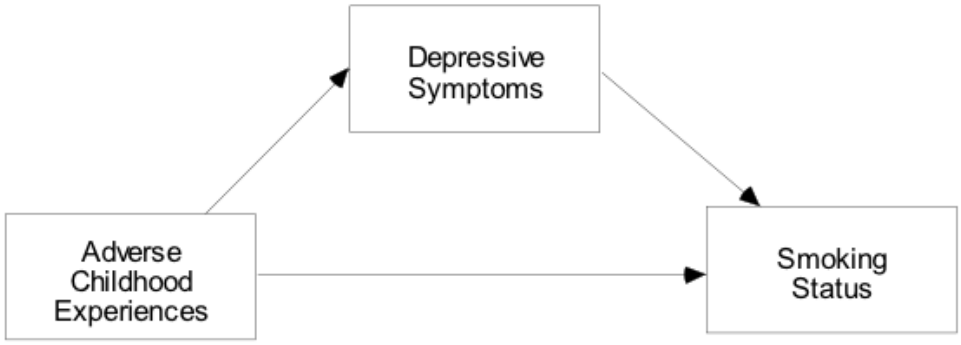


Figure 2: Proposed Path Model with Labels

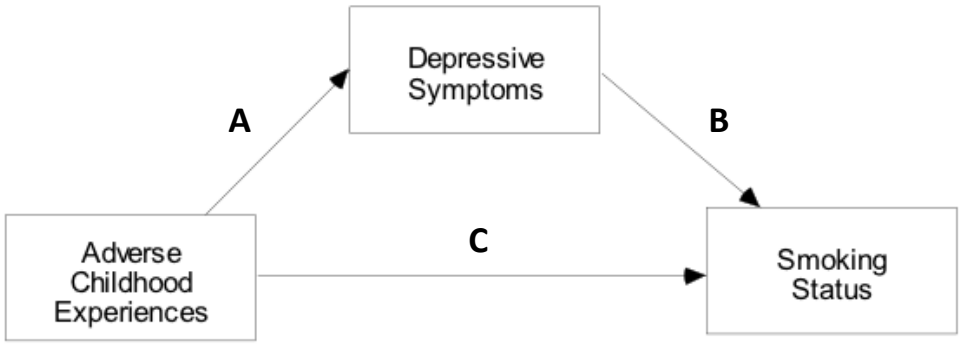
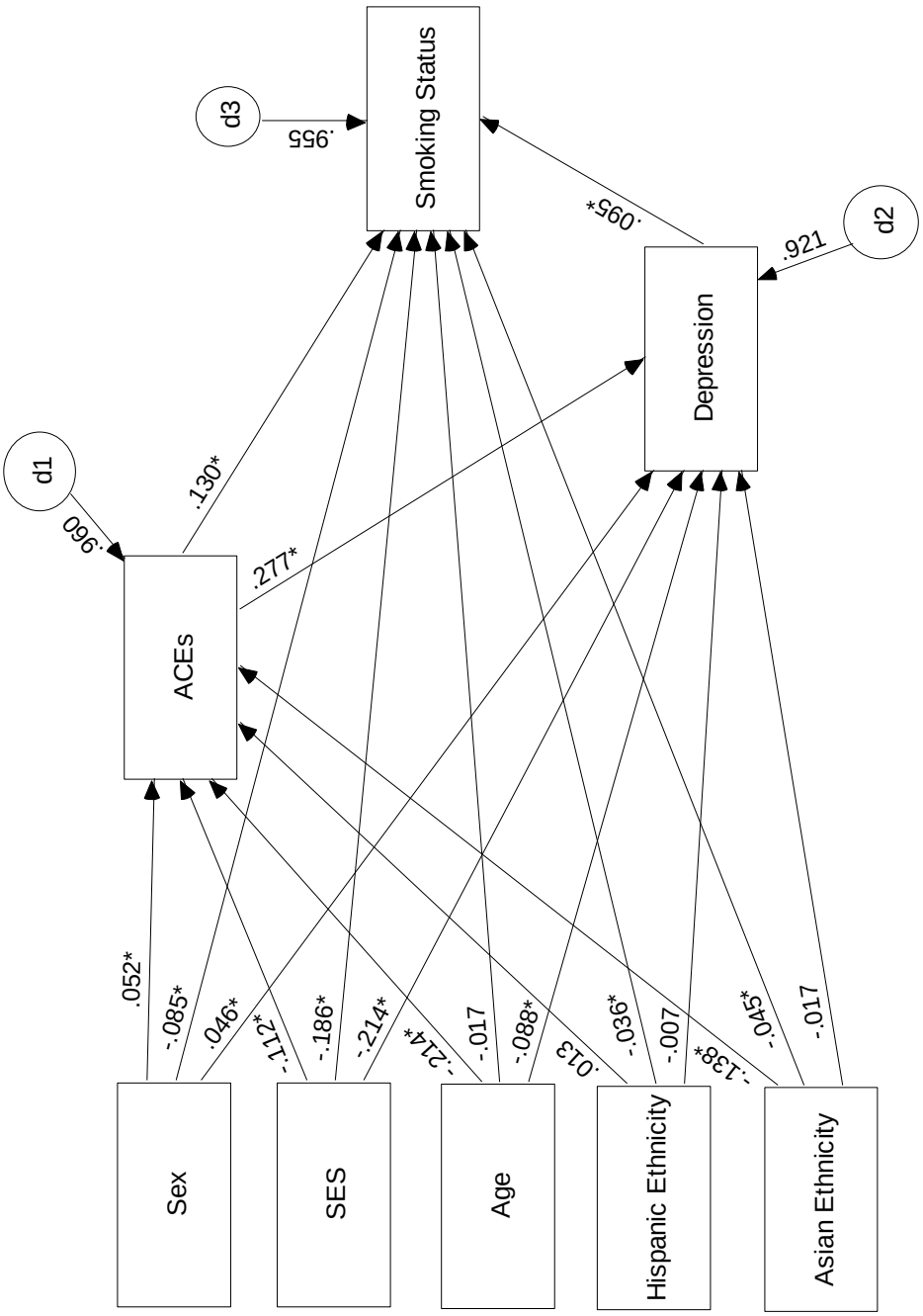


Figure 3: Path Model for White/Asian/Hispanic Subsample



Note: * $p < .01$

Appendix B: Tables

Table 1: Demographic Characteristics of Study Sample

Demographic Characteristics of Study Sample					
State	% female (sample)	% female (pop.)	% White (sample)	% White (pop.)	Mean Age (sample)
Hawaii	57.19	50.10	26.28	27.18	56.73
Nevada	57.10	49.36	74.39	59.80	55.34
Vermont	58.15	51.35	96.21	95.78	56.68
Wisconsin	57.14	50.73	91.60	87.47	55.29
United States	---	50.80	---	63.70	---

Table 2: Correlations between Study Variables, Full Sample

Correlations Between Study Variables, Full Sample						
	Variables	1	2	3	4	5
1	Age	--				
2	Sex	.004	--			
3	SES	-.128*	-.058*	--		
4	ACE Score	-.214*	.050*	-.092*	--	
5	Depressive Symptoms	-.115*	.073*	-.230*	.323*	--
6	Smoking Status	-.030*	-.060*	-.217*	.186*	.181*
Note: * $p < .01$						

Table 3: Prevalence of Adverse Childhood Experiences by Demographic Characteristics

Prevalence of Adverse Childhood Experiences by Demographic Characteristics							
		% reporting number of ACE categories					
Characteristic	Sample (N)	0	1	2	3	4 +	Mean Score
Gender							
Female	11,607	43.6	21.7	12.3	8.1	14.2	1.42 (1.78)
Male	7,898	44.9	22.8	12.9	8.1	11.3	1.28 (1.62)
Age group							
18-24	622	33.8	23.0	13.7	9.6	19.9	1.81 (1.97)
25-34	1,429	34.0	21.7	13.2	8.7	22.4	1.95 (2.13)
35-44	2,479	36.2	21.4	13.0	9.9	19.5	1.74 (1.91)
45-54	3,905	37.3	21.9	13.6	10.3	16.8	1.64 (1.83)
55-64	4,891	43.9	22.1	13.7	7.8	12.6	1.33 (1.65)
65 and older	6,179	55.2	22.7	10.5	6.0	5.7	.88 (1.31)
Ethnicity							
White	13,971	43.3	22.5	12.7	8.2	13.1	1.37 (1.71)
Black	520	32.5	24.0	15.8	9.6	18.1	1.77 (1.89)
Asian	2,096	64.1	20.2	8.8	3.8	3.1	.63 (1.08)
Native Hawaiian or Pacific Islander	157	36.3	26.8	9.6	9.6	17.8	1.61 (1.80)
American Indian or Alaskan Native	157	29.3	12.1	18.5	13.4	26.8	2.28 (2.12)
Other (non-Hispanic)	142	45.8	19.0	9.9	9.2	16.1	1.48 (1.87)
Multiracial	1,488	33.2	21.7	14.4	10.9	19.9	1.83 (1.91)
Hispanic	725	35.4	21.4	13.5	10.3	19.3	1.80 (2.00)

Table 4: Depressive Symptoms by Demographic Characteristics

Depressive Symptoms by Demographic Characteristics		
Characteristic	Sample size	Mean PHQ-8 score
Gender		
Female	11,634	3.26 (4.23)
Male	7,881	2.66 (3.89)
Age group		
18-24	627	3.64 (3.84)
25-34	1,441	3.58 (4.41)
35-44	2,530	3.57 (4.49)
45-54	3,990	3.48 (4.60)
55-64	4,936	2.90 (4.13)
65 and older	5,991	2.37 (3.33)
Ethnicity		
White	14,025	2.98 (4.02)
Black	529	4.61 (5.46)
Asian	2,048	2.11 (3.19)
Native Hawaiian or Pacific Islander	157	3.08 (4.09)
American Indian or Alaskan Native	158	5.52 (6.67)
Other (non-Hispanic)	137	3.42 (4.25)
Multiracial	1,496	3.44 (4.45)
Hispanic	735	3.69 (4.34)

Table 5: Smoking Status by Demographic Characteristics

Smoking Status by Demographic Characteristics					
Characteristic	Sample size (n)	Never smoked	Former smoker	Current, some days	Current, everyday
Gender					
Female	12,292	55.5	29.9	4.0	10.6
Male	8,351	44.8	39.8	4.1	11.4
Age group					
18-24	645	75.8	6.5	5.3	12.4
25-34	1,482	54.9	20.3	7.4	17.4
35-44	2,613	59.2	22.3	5.5	13.1
45-54	4,126	54.1	27.4	4.9	13.6
55-64	5,177	47.4	37.4	3.9	11.3
65 and older	6,600	45.9	45.5	2.1	6.4
Ethnicity					
White	14,762	49.3	36.2	3.9	10.6
Black	567	52.7	24.7	7.8	14.8
Asian	2,190	64.8	26.8	2.1	6.2
Native Hawaiian or Pacific Islander	165	52.1	27.3	4.8	15.8
American Indian or Alaskan Native	170	34.1	30.6	9.4	25.9
Other	150	46.7	33.3	6.7	13.3
Multiracial	1,582	49.6	29.0	5.4	16.0
Hispanic	780	56.7	26.8	4.9	11.7

Table 6: Regression Results for Smoking Status, Full Sample

Regression Results for Smoking Status, Full Sample				
Variable	*B*	SE *B*	*β*	*p*
SES	-.229	.008	-.210	<.001
Sex	-.160	.014	-.082	<.001
Age	-.002	.000	-.025	.001
D1 (Black)	-.032	.044	-.005	.474
D2 (Asian)	-.138	.023	-.045	<.001
D3 (Hawaiian)	-.059	.077	-.006	.445
D4 (American Indian)	.303	.079	.028	<.001
D5 (Other)	.061	.083	.005	.461
D6 (Multiracial)	.056	.027	.016	.035
D7 (Hispanic)	-.166	.037	-.033	<.001
ACE Score	.088	.004	.158	<.001
Note: $R^2 = .086$, $p < .001$				

Table 7: Simultaneous Regression Results for Smoking Status, White/Asian/Hispanic Subsample

Simultaneous Regression Results for Smoking Status, White/Asian/Hispanic Subsample				
Variable	*B*	SE *B*	*β*	*p*
SES	-.203	.009	-.186	<.001
Sex	-.162	.015	-.085	<.001
Age	-.001	.001	-.017	.049
D1 (Asian)	-.127	.023	-.045	<.001
D2 (Hispanic)	-.163	.037	-.036	<.001
ACE Score	.072	.005	.130	<.001
Depressive Symptoms	.023	.002	.095	<.001
Note: $R^2 = .088$, $p < .001$				

Table 8: Simultaneous Regression Results for Depressive Symptoms, White/Asian/Hispanic Subsample

Simultaneous Regression Results for Depressive Symptoms, White/Asian/Hispanic Subsample				
Variable	*B*	SE *B*	*β*	*p*
SES	-.982	.036	-.214	<.001
Sex	.365	.062	.046	<.001
Age	-.023	.002	-.088	<.001
D1 (Asian)	-.201	.093	-.017	.031
D2 (Hispanic)	-.143	.150	-.007	.341
ACE Score	.645	.019	.277	<.001
Note: $R^2 = .151$, $p < .001$				

Table 9: Simultaneous Regression Results for ACE Score, White/Asian/Hispanic Subsample

Simultaneous Regression Results for ACE Score, White/Asian/Hispanic Subsample				
Variable	*B*	SE *B*	*β*	*p*
SES	-.221	.016	-.112	<.001
Sex	.179	.028	.052	<.001
Age	-.024	.001	-.214	<.001
D1 (Asian)	-.710	.041	-.138	<.001
D2 (Hispanic)	.110	.067	.013	.102
Note: $R^2 = .079$, $p < .001$				

Table 10: Sequential Regression Results for Smoking Status, White/Asian/Hispanic Subsample

Sequential Regression Results for Smoking Status, White/Asian/Hispanic Subsample												
	Model 1				*Model 2*				*Model 3*			
Variable	*B*	SE *B*	*β*	*p*	*B*	SE *B*	*β*	*p*	*B*	SE *B*	*β*	*p*
SES	-.243	.009	-.223	<.001	-.224	.009	-.205	<.001	-.202	.009	-.185	<.001
Sex	-.144	.015	-.075	<.001	-.159	.015	-.083	<.001	-.168	.015	-.087	<.001
Age	-.003	.000	-.052	<.001	-.001	.001	-.019	.024	-.001	.001	-.010	.205
D1 (Asian)	-.193	.023	-.067	<.001	-.132	.023	-.045	<.001	-.127	.023	-.044	<.001
D2 (Hispanic)	-.152	.038	-.033	<.001	-.161	.037	-.034	<.001	-.157	.037	-.034	<.001
ACE Score					.086	.005	.155	<.001	.071	.005	.129	<.001
Depressive Symptoms									.022	.002	.093	<.001
R^2	.058				.080				.087			
F change	181.834				354.454				119.255			
p of *F* change	<.001				<.001				<.001			

Table 11: Direct, Indirect, and Total Standardized Effects on Smoking Status, White/Asian/Hispanic Subsample

Direct, Indirect, and Total Standardized Effects on Smoking Status, White/Asian/Hispanic Subsample

Variable	Direct	Indirect	Total
Depressive Symptoms	.095	—	.095
ACE Score	.130	.025	.155
Socioeconomic Status	-.186	-.037	-.223
Sex	-.085	.010	-.075
Age	-.017	-.035	-.052
Asian Ethnicity	-.045	-.022	-.067
Hispanic Ethnicity	-.036	.003	-.033

Table 12: Simultaneous Regression Results for Smoking Status, Black Subsample

Simultaneous Regression Results for Smoking Status, Black Subsample				
Variable	*B*	SE *B*	*β*	*p*
SES	-.209	.059	-.169	<.001
Sex	-.348	.109	-.148	.002
Age	.009	.003	.129	.006
ACE Score	.053	.029	.091	.073
Depressive Symptoms	.016	.010	.079	.121
Note: $R^2 = .086$, $p < .001$				

Table 13: Sequential Regression Results for Smoking Status, Black Subsample

Sequential Regression Results for Smoking Status, Black Subsample												
	Model 1				*Model 2*				*Model 3*			
Variable	*B*	SE *B*	*β*	*p*	*B*	SE *B*	*β*	*p*	*B*	SE *B*	*β*	*p*
SES	-.249	.057	-.202	<.001	-.229	.057	-.186	<.001	-.209	.059	-.169	<.001
Sex	-.330	.108	-.141	.002	-.323	.108	-.137	.003	-.348	.109	-.148	.002
Age	.007	.003	.098	.034	.009	.003	.127	.008	.009	.003	.129	.205
ACE Score					.068	.028	.116	.015	.053	.029	.091	.073
Depressive Symptoms									.016	.010	.079	.121
R^2	.068				.081				.086			
F change	10.744				5.935				2.413			
p of *F* change	<.001				.015				.121			

Table 14: Regression Results for Depressive Symptoms, Black Subsample

Regression Results for Depressive Symptoms, Black Subsample				
Variable	*B*	SE *B*	*β*	*p*
SES	-1.276	.265	-.209	<.001
Sex	1.542	.499	.133	.002
Age	-.011	.015	-.032	.464
ACE Score	.936	.129	.325	<.001
Note: R^2 =.193 , $p < .001$				

Table 15: Regression Results for ACE Score, Black Subsample

Regression Results for ACE Score, Black Subsample				
Variable	*B*	SE *B*	*β*	*p*
SES	-.300	.097	-.142	.002
Sex	-.110	.185	-.027	.553
Age	-.030	.006	-.249	<.001
Note: $R^2 = .080$, $p < .001$				

Appendix C: ACE Questions and Response Categories

ACE Questions and Response Categories			
ACE Category	Question	Response Options	Criterion
Mentally ill household member	Did you live with anyone who was depressed, mentally ill, or suicidal?	Yes/No	Yes
Household substance abuse	Did you live with anyone who was a problem drinker or alcoholic? Did you live with anyone who used illegal street drugs or who abused prescription medications?	Yes/No Yes/No	Yes (to either or both questions)
Incarceration of household member	Did you live with anyone who served time or was sentences to serve time in a prison, jail, or other correctional facility?	Yes/No	Yes
Parental divorce or separation	Were your parents separated or divorced?	Yes/No	Yes
Domestic violence	How often did your parents or adults in your home ever slap, hit, kick, punch or beat each other up?	Never, Once, More than once	Once or More than once
Physical abuse	Before age 18, how often did a parent or adult in your home ever hit, beat, kick, or physically hurt you in any way? Do not include spanking. Would you say---	Never, Once, More than once	Once or More than once
Emotional	How often did a parent or adult	Never, Once,	More than

abuse	in your home ever swear at you, insult you, or put you down?	More than once	once
Sexual abuse	How often did anyone 5 years older than you or an adult ever touch you sexually?	Never, Once, More than once	Once or more than once to any question
	How often did anyone at least 5 years older than you or an adult try to make you touch them sexually?	Never, Once, More than once	
	How often did anyone 5 years older than you or an adult force you to have sex?	Never, Once, More than once	

References

Aaron, L., & Dallaire, D. (2010). Parental incarceration and multiple risk experiences: Effects on family dynamics and children's delinquency. *Journal of Youth & Adolescence, 39*(12), 1471-1484.

Afifi, T. O., Enns, M. W., Cox, B. J., Asmundson, G. J., Stein, M. B., & Sareen, J. (2008). Population attributable fractions of psychiatric disorders and suicide ideation and attempts associated with adverse childhood experiences. *American Journal of Public Health*, *98*(5), 946-952.

Amato, P. R. (2001). Children of divorce in the 1990s: An update of the Amato and Keith (1991) meta-analysis. *Journal of Family Psychology, 15*(3), 355-370.

Amato, P. R. (2010). Research on divorce: Continuing trends and new developments. *Journal of Marriage and Family, 72*(3), 650-666.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Ammerman, R. T., Kolko, D. J., Kirisci, L., Blackson, T. C., & Dawes, M. A. (1999). Child abuse potential in parents with histories of substance use disorder. *Child Abuse & Neglect*, *23*(12), 1225-1238.

Anda, R. F., Croft, J. B., Felitti, V. J., Nordenberg, D., Giles, W. H., Williamson, D. F., & Giovino, G. A. (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA, 282*(17), 1652-1658.

Anda, R. F., Brown, D. W., Felitti, V. J., Bremner, J., Dube, S. R., & Giles, W. H. (2007). Adverse childhood experiences and prescribed psychotropic medications in adults. *American Journal Of Preventive Medicine, 32*(5), 389-394.

Anda, R. F., Butchart, A. & Felitti, V. J., & Brown, D. W. (2010). Building a framework for global surveillance of the public health implications of adverse childhood experiences. *American Journal of Preventive Medicine 39*(1), 93-98.

Anda, R. F., Williamson, D. F., Escobedo, L. G., Mast, E. E., Giovino, G. A., & Remington, P. L. (1990). Depression and the dynamics of smoking: A national perspective. *JAMA, 264*, 1541-1545.

Antoni, M. H., Lutgendorf, S. K., Cole, S. W., Dhabhar, F. S., Sephton, S. E., McDonald, P. G., Stefanek, M., & Sood, A. K. (2006). The influence of bio-behavioural factors on tumour biology: pathways and mechanisms. *Nature Reviews Cancer*, *6*(3), 240-248.

Appel, A. E. & Holden, G. W. (1998). The co-occurrence of spouse and physical child abuse: A review and appraisal. *Journal Of Family Psychology*, *12*(4), 578-599.

Arriola, K. R., Louden, T., Doldren, M. A., & Fortenberry, R. M. (2005). A meta-analysis of the relationship of child sexual abuse to HIV risk behavior among women. *Child Abuse & Neglect, 29*(6), 725-46.

Audrain-McGovern, J., Rodriguez, D., Rodgers, K., & Cuevas J. (2010). Declining alternative reinforcers link depression to young adult smoking. *Addiction, 106*(1), 178-187.

Bandura, A. (1977). *Social learning theory*. Englewood Cliffs, NJ: Prentice-Hall.

Banyard, V. L., Williams, L. M., Saunders, B. E., & Fitzgerald, M. M. (2008). The complexity of trauma types in the lives of women in families referred for family violence: multiple mediators of mental health. *American Journal Of Orthopsychiatry, 78*(4), 394-404.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173–1182.

Baxter, J., Weston, R., & Qu, L. (2011). Family structure, co-parental relationship quality, post-separation paternal involvement and children's emotional wellbeing. *Journal of Family Studies, 17*(2), 86-109.

Beck, A.T. (1967). *Depression: Clinical, experimental, and theoretical aspects.* New York: Hoeber.

Beck, A. T. (1976) *Cognitive therapy and the emotional disorders*. New York: Penguin Books.

Beck, C. T. (1999). Maternal depression and child behaviour problems: a meta-analysis. *Journal of Advanced Nursing, 29*(3), 623–629. doi: 10.1046/j.1365-2648.1999.00943.x

Black, P., & Garbutt, L. (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research*, *52*, 1– 23.

Bocknek, E., Sanderson, J., & Britner, P. (2009). Ambiguous loss and posttraumatic stress in school-age children of prisoners. *Journal Of Child & Family Studies, 18*(3), 323-333.

Boyd, S. J., Plemons, B. W., Schwartz, R. P., Johnson, J. L., & Pickens, R. W. (1999). The relationship between parental history and substance use severity in drug treatment patients. *The American Journal on Addictions, 8*(1), 15–23.

Breslau, N., Peterson, E. L., Schultz, L. R., Chilcoat, H. D., & Andreski, P. (1998). Major depression and stages of smoking: A longitudinal investigation. *Archives of General Psychiatry*, *55*(2),161-166.

Briere, J. & Runtz, M. (1990). Differential adult symptomatology associated with three types of child abuse histories. *Child Abuse & Neglect, 14*(3), 357–364.

Bronfenbrenner, U. (1979). *The ecology of human development*. Cambridge, MA: Harvard University Press.

Brook, J. S., Cohen, P., & Brook, D. W. (1998). Longitudinal study of co-occurring psychiatric disorders and substance use. *Journal of American Academy of Child & Adolescent Psychiatry, 37*(3), 322-30.

Brown, D. W., Anda, R. F., Tiemeier, H., Felitti, V. J., Edwards, V. J., Croft, J. B., & Giles, W.H. (2009). Adverse childhood experiences and the risk of premature mortality. *American Journal Of Preventive Medicine, 37*(5), 389-396.

Brown, D. W., Anda, R. F., Felitti, V. J., Edwards, V. J., Malarcher, A. M., Croft, J. B., & Giles, W. H. (2010). Adverse childhood experiences are associated with the risk of lung cancer: a prospective cohort study. *BMC Public Health, 10*, 20.

Brown, J., Cohen, P., Johnson, J. G. & Smailes, E. M. (1999). Childhood abuse and neglect: Specificity of effects on adolescent and young adult depression and suicidality. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(12), 1490-1496.

Brown, R. A., Lewinsohn, P. M., Seeley, J. R., & Wagner, E. F. (1996). Cigarette smoking, major depression, and other psychiatric disorders among adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*(12), 1602–1610.

Brown, S. A. (2008). Prevalence of alcohol and drug involvement during childhood and

adolescence. In Beauchaine, T. P. and Hinshaw, S. P. (Eds.) *Child and Adolescent Psychopathology* (pp. 405-444). Wiley: Hoboken, NJ.

Browne, C., & Winkelman, C. (2007). The effect of childhood trauma on later psychological adjustment. *Journal of Interpersonal Violence, 22*(6), 684-697.

Buser, T. J. & Hackney, H. (2012). Explanatory style as a mediator between childhood emotional abuse and nonsuicidal self-injury. *Journal Of Mental Health Counseling*, *34*(2), 154-169.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*(5582), 851-854.

Centers for Disease Control and Prevention (2004). Behavioral Risk Factor Surveillance System 2003 year-to-date data quality report handbook version 3.0.0. Available at http://www.cdc.gov/brfss/technical_infodata/pdf/2003DQRHandbook.pdf. (Accessed 30 April 2012).

Centers for Disease Control and Prevention. (2006). Behavioral Risk Factor Surveillance System operational and user's guide version 3.0. Available at ftp://ftp.cdc.gov/pub/

Data/Brfss/userguide.pdf (Accessed 21 April 2012).

Centers for Disease Control and Prevention. (2008). Smoking-Attributable Mortality, Years ofPotential Life Lost, and Productivity Losses – United States, 2000–2004. *Morbidity and Mortality Weekly Report*, *57*(45), 1226–28.

Centers for Disease Control and Prevention (CDC). (2010a). *Behavioral Risk Factor*

Surveillance System Survey Data [Data file]. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Available from: http://www.cdc.gov/brfss/technical_infodata/surveydata/2010.htm.

Centers for Disease Control and Prevention (CDC). (2010b). *Behavioral Risk Factor Surveillance System survey questionnaire*. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Available from: http://www.cdc.gov/brfss/questionnaires/english.htm.

Centers for Disease Control and Prevention (CDC). (2010c). BRFSS policy memo 97.1:

Cognitive testing. Retrieved from: http://www.cdc.gov/brfss/technical_infodata/

memos/971.htm. (Accessed 21 April 2012). Centers for Disease Control and Prevention (CDC). (2010d). *Behavioral Risk Factor Surveillance System codebook*. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Available from: http://www.cdc.gov/brfss/questionnaires/english.htm.

Centers for Disease Control and Prevention (2011a). Behavioral Risk Factor Surveillance System 2010 summary data quality report (Version #1). Available at http://www.cdc.gov/brfss/technical_infodata/quality.htm. (Accessed 21 April 2012).

Centers for Disease Control and Prevention (2011b). 2010 BRFSS overview. Available from: http://www.cdc.gov/brfss/technical_infodata/surveydata/2010.htm. (Accessed 1 May 2012).

Centers for Disease Control and Prevention. (2012). Adult cigarette smoking in the United States: Current estimate. *Fact Sheets: Smoking and Tobacco Use.* Retrieved from: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/adult_data/cig_smoking/

index.htm. (Accessed 11 February 2012).

Chaiton, M., Cohen, J., O'Loughlin, J., & Rehm, J. (2010). Use of cigarettes to improve affect and depressive symptoms in a longitudinal study of adolescents. *Addictive Behaviors, 35*(12), 1054-60.

Chapman, D. P., Anda, R. F., Felitti, V. J., Dube, S. R., Edwards, V. J., & Whitfield, C. L. (2004). Adverse childhood experiences and the risk of depressive disorders in adulthood. *Journal of Affective Disorders 82*(2), 217–225.

Chatterji , P., & Markowitz, S. (2001). The impact of maternal alcohol and illicit drug use on children's behavior problems: Evidence from the children of the National Longitudinal Survey of Youth. *Journal of Health Economics, 20*(5), 703-731.

Chen, L. P., Murad, M. H., Paras, M. L., Colbenson, K. M., Sattler, A. L., Goranson, E. N., Elamin, M. B., Seime, R. J., Shinozaki, G., Prokop, L. J., & Zirakzadeh, A. (2010). Sexual abuse and lifetime diagnosis of psychiatric disorders: systematic review and meta-analysis. *Mayo Clinic Proceedings, 85*(7), 618–629.

Christensen, H. B., & Bilenberg, N. (2000). Behavioral and emotional problems in children of alcoholic mothers and fathers. *European Child & Adolescent Psychiatry*, *9*(3), 219-226.

Cicchetti, D. & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry*, *53*, 96-118.

Cicchetti, D. (2006). Developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 1: Theory and method* (2nd ed., pp. 1-23). New York: Wiley.

Cicchetti, D. (2008). A multiple-levels-of-analysis perspective on research in development and psychopathology. In Beauchaine, T. P. and Hinshaw, S. P. (Eds.) *Child and Adolescent Psychopathology* (pp. 27-57). Wiley: Hoboken, NJ.

Claussen, A. H., & Crittenden, P. M. (1991). Physical and psychological maltreatment: Relations among types of maltreatment. *Child Abuse & Neglect, 15*(1), 5-18.

Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *JAMA*, *298*(14), 1685-1687.

Cohen, S. & Williamson, G. M. (1991). Stress and infectious disease in humans. *Psychological Bulletin, 109*, 5–24.

Cole, S. W., Kemeny, M. E., Fahey, J. L., Zack, J. A., & Naliboff, B. D. (2003). Psychological risk factors for HIV pathogenesis: mediation by the autonomic nervous system. *Biological Psychiatry*, *54*(12), 1444-1456.

Covey, L. S., Glassman, A. H., Stetner, F., & Becker, J. (1993). Effect of history of alcoholism or major depression on smoking cessation. *American Journal of Psychiatry, 150*, 1546-1547.

Cyranowski, J. M., Schott, L. L., Kravitz, H. M., Brown, C., Thurston, R. C., Joffe, H., ... & Bromberger, J. T. (2012). Psychosocial features associated with lifetime comorbidity of major depression and anxiety disorders among a community sample of mid- life women: the swan mental health study. *Depression and Anxiety*, 29(12), 1050–1057.

Dallaire, D. H. & Aaron, L. (2010). Middle childhood: family, school, and peer contexts for children affected by parental incarceration. In J.M. Eddy & J. Poehlmann

(Eds.), *Children of Incarcerated Parents: A Handbook for Researchers and Practitioners* (pp. 101 - 119). Washington, D.C: The Urban Institute Press.

De Bellis, M. D., Broussard, E. R., Herring, D. J., Wexler, S., Moritz, G., & Benitez, J. (2001). Psychiatric co-morbidity in caregivers and children involved in maltreatment: A pilot research study with policy implications. *Child Abuse & Neglect, 25*(7), 923-944.

Della Femina, D., Yeager, C. A., & Lewis, D. O. (1990). Child abuse: adolescent records vs. adult recall. *Child Abuse & Neglect, 14*(2), 227–231.

Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., Loo, C. M., & Giles, W. H. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect, 28*(7), 771-784.

Douglas, K. R., Chan, G., Gelernter, J., Arias, A. J., Anton, R. F., Weiss, R. D., Brady, K., Poling, J., Farrer, L., & Kranzler, H. R. (2010). Adverse childhood events as risk factors for substance dependence: Partial mediation by mood and anxiety disorders. *Addictive Behaviors 35*(1), 7–13.

Downs, W. R., Capshew, T., & Brindels, B. (2004). Relationships between adult women's alcohol problems and their childhood experiences of parental violence and psychological aggression. *Journal of Studies on Alcohol, 65*(3), 336-345.

Dozois, D. J. A. (2002). Cognitive organization of self-schematic content in nondysphoric, mildly dysphoric, and moderately-severely dysphoric individuals. *Cognitive Therapy and Research, 26*(3), 417–429.

Dozois, D. J. A. (2007). Stability of negative self-structures: A longitudinal comparison of depressed, remitted, and nonpsychiatric controls. *Journal of Clinical Psychology, 63*(4), 319–338.

Dozois, D. J. A., & Beck, A. T. (2008). Cognitive schemas, beliefs, and assumptions. In K. S. Dobson & D. J. A. Dozois (Eds.), *Risk factors in depression*. Oxford, England: Elsevier.

Dube, S. R., Williamson, D. F., Thompson, T., Felitti, V. J. & Anda, R. F. (2004). Assessing the reliability of retrospective reports of adverse childhood experiences among adult HMO members attending a primary care clinic. *Child Abuse & Neglect, 28*, 729-737.

Duijts, S. F. A., Zeegers, M. P. A., & Borne, B. V. (2003). The association between stressful life events and breast cancer risk: a meta-analysis. *International Journal of Cancer*, *107*(6), 1023-1029.

Edwards, V. J., Anda, R. F., Gu, D., Dube, S. R., & Felitti VJ. (2007). Adverse childhood experiences and smoking persistence in adults with smoking-related symptoms and illness. *Permanente Journal, 11*, 5–7.

Engel, G. L. (1977). The need for a new medical model: a challenge for biomedicine. *Science, 196*, 129–36.

Engel, G. L. (1997). From biomedical to biopsychosocial: being scientific in the human domain. *Psychosomatics, 38*(6), 521-528.

Ershler, W. B. & Keller, E. T. (2000). Age-associated increased interleukin-6 gene expression, late-life diseases, and frailty. *Annual Review of Medicine, 51*, 245–270.

Escobedo, L. G., Reddy, M., & Giovino, G. A. (1998). The relationship between depressive symptoms and cigarette smoking in U.S. adolescents. *Addiction, 93*(3), 433–440.

Evans, E., Hawton, K. & Rodham, K. (2005). Suicidal phenomena and abuse in adolescents: A review of epidemiological studies. *Child Abuse & Neglect, 29*(1), 45–58.

Evans, S. E., Davies, C., & DiLillo, D. (2008). Exposure to domestic violence: A meta-analysis of child and adolescent outcomes. *Aggression & Violent Behavior*, *13*(2), 131-140.

Famularo, R. Kinscherff, R. & Fenton, T. (1992). Psychiatric diagnoses of abusive mothers: A preliminary report. *Journal of Nervous and Mental Disease, 180*(10), 658-661.

Felitti, V.J., Anda, R.F., Nordenberg, D., Williamson, D.F., Spitz, A.M., Edwards, V., Koss, M.P., & Marks, J.S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the Adverse Childhood Experiences (ACE) study. *American Journal of Preventive Medicine, 14*, 245– 258.

Ferdinand, R. F., Blum, M., & Verhulst, F. C. (2001). Psychopathology in adolescence predicts substance use in young adulthood. *Addiction, 96*(6), 861–870.

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2008). Exposure to childhood sexual and physical abuse and adjustment in early adulthood. *Child Abuse & Neglect, 32*(6), 607-619.

Finkelhor, D. Ormrod, R. K. & Turner, H. A. (2007). Poly-victimization: a neglected component in child victimization. *Child Abuse & Neglect, 31*(1), 7-26.

Fishbein, D., Novak, S. P., Krebs, C., Warner, T., & Hammond J. (2011). The mediating effect of depressive symptoms on the relationship between traumatic childhood experiences and drug use initiation. *Addictive Behaviors, 36*(5), 527–531.

Fleiss, J. L. (1981). *Statistical methods for rates and proportions* (2nd ed.). New York: Wiley.

Foley, D. L., Neale, M. C., & Kendler, K. S. (2001). Genetic and environmental risk factors for depression assessed by subject rated symptom check list versus structured clinical interview. *Psychological Medicine, 31*(8), 1413–1423.

Ford, E. S., Anda, R. F., Edwards, V. J., Perry, G. S., Zhao, G., Tsai, J., Li, C., & Croft, J. B. (2011). Adverse childhood experiences and smoking status in five states. *Preventive Medicine, 53*(3): 188-93.

Freisthler, B. (2011). Alcohol use, drinking venue utilization, and child physical abuse: Results from a pilot study. *Journal of Family Violence, 26*(3), 185-193.

Friedman-Wheeler, D. G., Ahrens, A. H., Haaga, D. A. F., McIntosh, E., & Thorndike, F. P. (2007). Depressive symptoms, depression proneness, and outcome expectancies for cigarette smoking. *Cognitive Therapy & Research*, *31*(4), 547-557.

Garrett, B. E., Dube, S. R., Trosclair, A., Caraballo, R. S., & Pechacek, T. F. (2011). Cigarette smoking - United States, 1965-2008. *Morbidity and Mortality Weekly Report Surveillance Summary, 60*(Supplement), 109-113.

Gibb, B. E., Alloy, L. B., Abramson, L. Y., Rose, D. T., Whitehouse, W. G., Donovan, P., … Tiernay, S. (2001). History of childhood maltreatment, depressogenic cognitive style, and episodes of depression in adulthood. *Cognitive Therapy and Research, 25*(4), 425–446.

Gilbert, R., Spatz Widom, C., Browne, K., Fergusson, D., Webb, E., & Janson, J. (2009). Burden and consequences of child maltreatment in high-income countries. *The Lancet, 373*, 68-81.

Glassman, A. H., Stetner, F., Walsh, B. T., Raizman, P. S., Fleiss, J. L., Cooper, T. B., & Covey, L. S. (1988). Heavy smokers, smoking cessation, and clonidine. *JAMA, 259,* 2863-2866.

Glaze, L. E., & Maruschak, L. M. (2008). *Parents in prison and their minor children* (NCJ 222984). Washington, DC: US Department of Justice, Bureau of Justice Statistics.

Hairston, C. F. (2010). Foreword. In J.M. Eddy & J. Poehlmann (Eds.), *Children of Incarcerated Parents: A Handbook for Researchers and Practitioners* (pp. xiii - xvi). Washington, D.C: The Urban Institute Press.

Hanlon, T. E., Blatchley, R. J., Bennett-Sears, T., O'Grady, K. E., Rose, M., & Callaman, J. M. (2005). Vulnerability of children of incarcerated addict mothers: Implications for preventive intervention. *Children & Youth Services Review, 27*(1), 67-84.

Hardt, J. and Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *Journal of Child Psychology and Psychiatry, 45*(2) 260–273.

Harkness, K. L., & Lumley, M. N. (2008). Child abuse and neglect and the development of depression in children and adolescents. In J. Abela & B. Hankin (Eds.), *Depression in children and adolescents* (pp. 639-657). New York: Guilford.

Harper, F. & Arias, I. (2004). The role of shame in predicting adult anger and depressive symptoms among victims of child psychological maltreatment. *Journal Of Family Violence, 19*(6), 359-367.

Herrenkohl, T. I. & Herrenkohl, R. C. (2007). Examining the overlap and prediction of multiple forms of child maltreatment, stressors, and socioeconomic status: A longitudinal analysis of youth outcomes. *Journal of Family Violence, 22*(7), 553–562.

Hinshaw, S. P. (2008). Developmental psychopathology as a scientific discipline: Relevance to behavioral and emotional disorders of childhood and adolescence. In Beauchaine, T. P. and Hinshaw, S. P. (Eds.) *Child and Adolescent Psychopathology* (pp. 3-26). Wiley: Hoboken, NJ.

Holt, M. K., Finkelhor, D., & Kantor, G. K. (2007). Multiple victimization experiences of urban elementary school students: Associations with psychosocial functioning and academic performance. *Child Abuse & Neglect, 31*(5), 503-515.

Huang, F.Y., Chung, H., Kroenke, K., Delucchi, K.L., Spitzer, R.L. (2006). Using the Patient Health Questionnaire-9 to measure depression among racially and ethnically diverse primary care patients. *Journal of General Internal Medicine 21*(6), 547–552.

Huang, Y., Hannon, P. A., Williams, B., & Harris, J. R. (2011). Workers' health risk behaviors by state, demographic characteristics, and health insurance status. *Prevention of Chronic Disease, 8*(1), A12. Retrieved from http://www.cdc.gov/pcd/issues/2011/jan/ 10_0017.htm.

Institute of Medicine (2003). *Unequal treatment: Confronting racial and ethnic disparities in health care.* Washington, D.C.: National Academy of Sciences.

Irish, L., Kobayashi, I. & Delahanty, D. L. (2010). Long-term physical health consequences of childhood sexual abuse: a meta-analytic review. *Journal of Pediatric Psychology, 35*(5), 450-461.

Johnson, R. M., Kotch, J. B., Catellier, D. J., Winsor, J. R., Dufort, V., Hunter, W., Amaya-Jackson, L. (2002). Adverse behavioral and emotional outcomes from child abuse and witnessed violence. *Child Maltreatment, 7*(3), 179-186.

Judd, L. L., Schettler, P. J., & Akiskal, H. S. (2002). The prevalence, clinical relevance, and public health significance of subthreshold depressions. *Psychiatric Clinics of North America, 25*(4), 685-698.

Kane, P. & Garber, J. (2004). The relations among depression in fathers, children's psychopathology, and father-child conflict: A meta-analysis. *Clinical Psychology Review, 24*(3), 339-360.

Keith, T. Z. (2006). *Multiple regression and beyond.* Boston: Allyn & Bacon.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., ... & Wang, P. S. (2003). The epidemiology of major depressive disorder. *JAMA: The Journal of the American Medical Association*, *289*(23), 3095-3105.

Kilpatrick, K. L., & Williams, L. M. (1998). Potential mediators of posttraumatic stress disorder in child witnesses to domestic violence. *Child Abuse & Neglect, 22*(4), 319-330.

King, G., Guilbert, P., Ward, D., Arwidson, P. & Noubary, F. (2006). Correlates of sexual abuse and smoking among French adults. *Child Abuse & Neglect, 30*(6), 709-723.

Kitzmann, K. M., Gaylord, N. K., Holt, A. R., Kenny, E. D. (2003). Child witnesses to domestic violence: a meta-analytic review. *Journal of Consulting & Clinical Psychology, 71*(2), 339-52.

Klein, D. N., Torpey, D. C., Bufferd, S. J., & Dyson, M. W. (2008). Depressive disorders. In Beauchaine, T. P. and Hinshaw, S. P. (Eds.), *Child and Adolescent Psychopathology* (pp. 477-509). Hoboken, NJ: Wiley.

Kroenke, K. & Spitzer, R. L. (2002). The PHQ-9: A new depression diagnostic and severity measure. *Psychiatric Annals*, *32*(9), 509-515.

Kroenke, K., Spitzer, R. L. & Williams, J. B. W. (2001). The PHQ-9. *Journal of General Internal Medicine, 16*, 606–613.

Kroenke, K., Strine, T. W., Spitzer, R. L., Williams, J. W., Berry, J. T., & Mokdad, A. H. (2009). The PHQ-8 as a measure of current depression in the general population. *Journal Of Affective Disorders*, *114*(1-3), 163-173.

Lahey, B. B. (2008). Oppositional defiant disorder, conduct disorder, and juvenile delinquency. In Beauchaine, T. P. and Hinshaw, S. P. (Eds.), *Child and Adolescent Psychopathology* (pp. 335-369). Hoboken, NJ: Wiley.

Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics, 33*(1), 159–174.

Lansford, J. E., Dodge, K. A., Pettit, G. S., Bates, J. E., Crozier, J. & Kaplow, J. (2002) A 12-year prospective study of the long-term effects of early child physical maltreatment on psychological, behavioral, and academic problems in adolescence. *Archives of Pediatrics and Adolescent Medicine, 156*(8), 824-830.

Lee, P.W., Schulberg, H.C., Raue, P.J., Kroenke, K. (2007). Concordance between the PHQ-9 and the HSCL-20 in depressed primary care patients. *Journal of Affective Disorders, 99*(1–3), 139–145.

Lehmann, P. (1997). The development of posttraumatic stress disorder (PTSD) in a sample of child witnesses to mother assault. *Journal of Family Violence, 12*(3), 241-257.

Lerman, C., Audrain, J., Orleans, C. T., Boyd, R., Gold, K., Main, D., & Caporaso, N. (1996). Investigation of mechanisms linking depressed mood to nicotine dependence. *Addictive Behaviors, 21*(1), 9-19.

Leserman, J., Petitto, J. M., Gu, H., Gaynes, B. N., Barroso, J., Golden, R. L., Perkins, D. O., Folds, J. D., & Evans, D. L. (2002). Progression to AIDS, a clinical AIDS condition and mortality: psychosocial and physiological predictors. *Psychological Medicine*, *32*(6), 1059-1073.

Leserman, J. (2005). Sexual abuse history: Prevalence, health effects, mediators and psychological treatment. *Psychosomatic Medicine, 67*(6), 906–915.

Lester, P. E., Weiss, R. E., Rice, E., Comulada, W. S., Lord, L., Alber, S. & Rotheram-Borus, M. J. (2009). The longitudinal impact of HIV+ parents' drug use on their adolescent children. *American Journal of Orthopsychiatry, 79*(1), 51–59.

Lewis, T., Kotch, J., Wiley, T., Litrownik, A.J., English, D., Thompson, R., Zolotor, A. J., Block, S. D., & Dubowitz, H. (2011). Internalizing problems: A potential pathway from child maltreatment to adolescent smoking. *Journal of Adolescent Health, 48*(3), 247-252.

Lovejoy, M. C., Graczyk, P. A., O'Hare, E., Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical Psychology Review, 20*(5), 561-592.

Lovibond, P. F. (1998). Long-term stability of depression, anxiety, and stress syndromes. *Journal of Abnormal Psychology, 107*(3), 520–526.

Löwe, B., Spitzer, R. L., Williams, J. B., Mussell, M., Schellberg, D., & Kroenke, K. (2008). Depression, anxiety and somatization in primary care: syndrome overlap and functional impairment. *General Hospital Psychiatry, 30*(3), 191-199.

Lumley, M. N., & Harkness, K. L. (2007). Specificity in the relations among childhood adversity, early maladaptive schemas, and symptom profiles in adolescent depression. *Cognitive Therapy & Research, 31*, 639–657.

Lumley, M. L., Harkness, K. L. (2009). Childhood maltreatment and depressotypic cognitive organization. *Cognitive Therapy & Research*, *33*(5), 511-522.

Margolin, G., Gordis, E. B., Medina, A. M., & Oliver, P. H. (2003). The co-occurrence of husband-to-wife aggression, family-of-origin aggression, and child abuse potential in a community sample: Implications for parenting. *Journal of Interpersonal Violence, 18*(4), 413-440.

McChargue, D. E., Spring, B, Cook, J. W., & Neumann, C. (2004). Reinforcement expectations explain the relationship between depressive history and smoking status in college students. *Addictive Behaviors, 29*(5), 991-994.

McFarlane, J., Groff, J., O'Brien, J., Watson, K. (2003). Behaviors of children who are exposed and not exposed to intimate partner violence: An analysis of 330 Black, White and Hispanic children. *Pediatrics, 112*(3), 202-207.

McKenzie, M., Olsson, C. A., Jorm, A. F., & Romaniuk, H., & Patton, G. C. (2010). Association of adolescent symptoms of depression and anxiety with daily smoking and nicotine dependence in young adulthood: findings from a 10-year longitudinal study. *Addiction, 105*(9), 1652-9.

Merikangas, K. R., Dierker, L. C., & Szatmari, P. (1998). Psychopathology among offspring of parents with substance abuse and/or anxiety disorders: A high-risk study. *Journal of Psychology and Psychiatry and Allied Disciplines, 39*(5), 711-720.

Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual Causes of Death in the United States, 2000. *JAMA*, *291*(10), 1238-1245.

Moonesinghe, R., Zhu, J., & Truman, B. I. (2011). Health Insurance Coverage – United States, 2004 and 2008. *Morbidity and Mortality Weekly Report, 60*, 35-37.

Moylan, C., Herrenkohl, T., Sousa, C., Tajima, E., Herrenkohl, R., & Russo, M. M. (2010). The effects of child abuse and exposure to domestic violence on adolescent internalizing and externalizing behavior problems. *Journal Of Family Violence*, *25*(1), 53-63.

Molnar, B. E., Buka, S. L., & Kessler, R. C. (2001). Child sexual abuse and subsequent psychopathology: Results from the National Comorbidity Survey. *American Journal of Public Health, 91*(5), 753–760.

Mustonen, U., Huurre, T., Haukkala, A., Kiviruusu, O., & Aro, H. (2011). Long-term impact of parental divorce on intimate relationship quality in adulthood and the mediating role of psychosocial resources. *Journal Of Family Psychology*, *25*(4), 615-619.

Nelson, D. E., Holtzman, D., Bolen, J., Stanwyck, C. A., & Mack, K. A. (2001). Reliability and validity of measures from the Behavioral Risk Factor Surveillance System (BRFSS). *Sozial- und Präventivmedizin/Social and Preventive Medicine, 46*(Suppl. 1), S03-44.

Nelson, D.E., Powell-Griner, E., Town, M., & Kovar, M. G. (2003). A comparison of national estimates from the National Health Interview Survey and the Behavioral Risk Factor Surveillance System. *American Journal of Public Health, 93*(8), 1335-1341.

Ney, P. G., Fung, T., & Wickett, A. R. (1994). The worst combinations of child abuse and neglect. *Child Abuse & Neglect, 18*(9), 705-14.

Nichols, H. B. & Harlow, B. L. (2004). Childhood abuse and risk of smoking onset. *Journal of Epidemiology and Community Health, 58*(5), 402-406.

Paolucci, E. O., Genuis, M. L., Violato, C. (2001). A meta-analysis of the published research on the effects of child sexual abuse. *Journal of Psychology, 135*(1), 17-36.

Perry, B. D. (2008). Child maltreatment: A neurodevelopmental perspective on the role of trauma and neglect in psychopathology. In Beauchaine, T. P. and Hinshaw, S. P. (Eds.), *Child and Adolescent Psychopathology* (pp. 93-128). Hoboken, NJ: Wiley.

Phillips, S. D., Barth, R. P., Burns, B. J., & Wagner, H. (2004). Parental arrest and children involved with child welfare services agencies. *American Journal Of Orthopsychiatry, 74*(2), 174-186.

Phillips, S. D., Erkanli, A., Keeler, G., Costello, E. J., & Angold, A. (2006). Disentangling the risks: Parental criminal justice involvement and children's exposure to family risks. *Criminology and Public Policy, 5*(4), 677–702.

Pinto-Meza, A., Serrano-Blanco, A., Penarrubia, M. T., Blanco, E., & Haro, J. M. (2005). Assessing depression in primary care with the PHQ-9: can it be carried out over the telephone? *Journal of General Internal Medicine 20*(8), 738–742.

Pomerleau, C. S., & Pomerleau, O. F. (1987). The effects of a psychological stressor on cigarette smoking and subsequent behavioral and physiological responses. *Psychophysiology, 24*(3), 278–285.

Prino, C. T. & Peyrot, M. (1994). The effect of child physical abuse and neglect on aggressive, withdrawn, and prosocial behavior. *Child Abuse & Neglect 18*(10), 871-884.

Read, J., Perry, B.D., Moskowitz, A., & Connolly, J. (2001). The contribution of early traumatic events to schizophrenia in some patients: A traumagenic neurodevelopmental model, *Psychiatry, 64*, 319–345.

Repetto, P. B., Caldwell, C. H., & Zimmerman, M. A. (2005). A longitudinal study of the relationship between depressive symptoms and cigarette use among African American adolescents. *Health Psychology, 24*(2), 209-219.

Rief, W., Nanke, A., Klaiberg, A., & Braehler, E. (2004). Base rates for panic and depression according to the Brief Patient Health Questionnaire: a population-based study. *Journal of Affective Disorders 82*(2), 271–276.

Riso, L. P., Miyatake, R. K., & Thase, M. E. (2002). The search for determinants of chronic depression: A review of six factors. *Journal of Affective Disorders, 70*(2), 103-116.

Salive, M. E., & Blazer, D. G. (1993). Depression and smoking cessation in older adults: A longitudinal study. *Journal of the American Geriatrics Society, 41,* 1313-1316.

Sameroff, A. J., Seifer, R., Baldwin, A. & Baldwin, C. (1993). Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development, 64*(1), 80-97.

Saunders, B. E. (2003). Understanding children exposed to violence: Toward an integration of overlapping fields. *Journal of Interpersonal Violence, 18*(4), 356-376.

Schleicher, H. E., Harris, K. J., Catley, D., & Nazir, N. (2009). The role of depression and negative affect regulation expectancies in tobacco smoking among college students. *Journal of American College Health, 57*(5), 507−512.

Schoenborn, C. A. (1995). Exposure to alcoholism in the family: United States, 1988. *Advance Data, 205, 1-13*. DHHS Publication No. (PHS)95-1880.

Segal, Z. V., Hood, J. E., Shaw, B. F., & Higgins, E. T. (1988). A structural analysis of the self-schema construct in major depression. *Cognitive Therapy and Research, 12*(5), 471–485.

Segerstrom, S. C. & Miller, G. E. (2004). Psychological stress and the human immune system: A meta-analytical study of 30 years of inquiry. *Psychological Bulletin, 130*(4), 601-630.

Simpson, T. L. & Miller, W. R. (2002). Concomitance between childhood sexual and physical abuse and substance use problems. A review. *Clinical Psychology Review,*

22(1), 27−77.

Smith, T. W. & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: current status and implications for research and practice. *Journal of Consulting & Clinical Psychology, 70*(3), 548-68.

Springer, K. W., Sheridan, J., Kuo, D., & Carnes, M. (2007). Long-term physical and mental health consequences of childhood physical abuse: Results from a large population-based sample of men and women. *Child Abuse & Neglect, 31*(5), 517-530.

Sternberg, K. J., Lamb, M. E., Greenbaum, C., Cicchetti, D., Dawud, S., Cortes, R. M., Krispin, O. & Lorey, F. (1993). Effects of domestic violence on children's behavior problems and depression. *Developmental Psychology, 29*(1), 44–52.

Sternberg, K. J., Lamb, M. E., Guterman, E., & Abbott, C. G. (2006). Effects of early and later family violence on children's behavior problems and depression: A longitudinal, multi-informant study. *Child Abuse & Neglect, 30*(10), 283−306.

Straus, M., & Gelles, R. J. (1990). *Physical violence in American families: Risk factors and adaptations to violence in 8,145 families.* New Brunswick: Transaction Press.

Sullivan, M., Carmody, D., & Lewis, M. (2010). How neglect and punitiveness influence emotion knowledge. *Child Psychiatry & Human Development*, *41*(3), 285-298.

Suls, J., & Rothman, A. (2004). Evolution of the biopsychosocial model: Prospects and challenges for health psychology. *Health Psychology*, *23*(2), 119-125.

Teicher, M. H., Andersen, S. L., Polcari, A., Andersen, C. M., & Navalta, C. P. (2002). Developmental neurobiology of childhood stress and trauma. *Psychiatric Clinics of North America, 25*(2), 397-426.

Thompson, M.P. Arias, I., Basile, K., & Desai, S. (2002). The association between childhood physical and sexual victimization and health problems in adulthood in a nationally representative sample of women. *Journal of Interpersonal Violence, 17*(10), 1115-1129.

Topitzes, J., Mersky, J. P., & Reynolds, A. J. (2010). Child maltreatment and adult cigarette smoking: A long-term developmental model. *Journal of Pediatric Psychology, 35*(5), 484-498.

Trice, A. D. & Brewster, J. (2004). The effects of maternal incarceration on adolescent children. *Journal of Police and Criminal Psychology, 19*(1), 27-35.

Turner, J. R. & Lloyd, D. A. (1995). Lifetime traumas and mental health: The significance of cumulative adversity. *Journal of Health and Social Behavior, 36*(4), 360–376.

Turner-Cobb, J. M., Sephton, S. E., & Speigel, D. (2001). Psychosocial effects on immune function and disease progression in cancer: human studies. In Ader, R., Felten, D. L., & Cohen, N. (Eds.), *Psychoneuroimmunology* (3rd ed.) (565-582). New York, NY: Academic Press.

Walker, E. A., Gelfand, A., Katon, W. J., Koss, M. P., Von Korff, M., Bernstein, D., & Russo, J. (1999). Adult health status of women with histories of childhood abuse and neglect. *American Journal of Medicine, 107*(4), 332-339.

Widom, C. S., DuMont, K., & Czaja, S. J. (2007). A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. *Archives of General Psychiatry, 64*(1), 49−56.

Williams, L. M. (1995). Recovered memories of abuse in women with documented child sexual victimization histories. *Journal of Trauma and Stress, 8*(4), 649–673.

Wilson, K., Hansen, D., & Li, M. (2011). The traumatic stress response in child maltreatment and resultant neuropsychological effects. *Aggression and Violent Behavior, 16*, 87-97.

Wu, L. T., & Anthony, J. C. (1999). Tobacco smoking and depressed mood in late childhood and early adolescence. *American Journal of Public Health*,*89*(12), 1837-1840.

Wyatt, G. E. (1985). The sexual abuse of Afro-American and White-American women in childhood. *Child Abuse & Neglect*, 9, 507–19.

Young, J. E., & Brown, G. (1994). *Young schema questionnaire–short form*. New York: Cognitive Therapy Center.

Young, J. E., Klosko, J. S., & Weishaar, M. E. (2003). *Schema therapy: A practitioner's guide*. New York: Guilford Press.